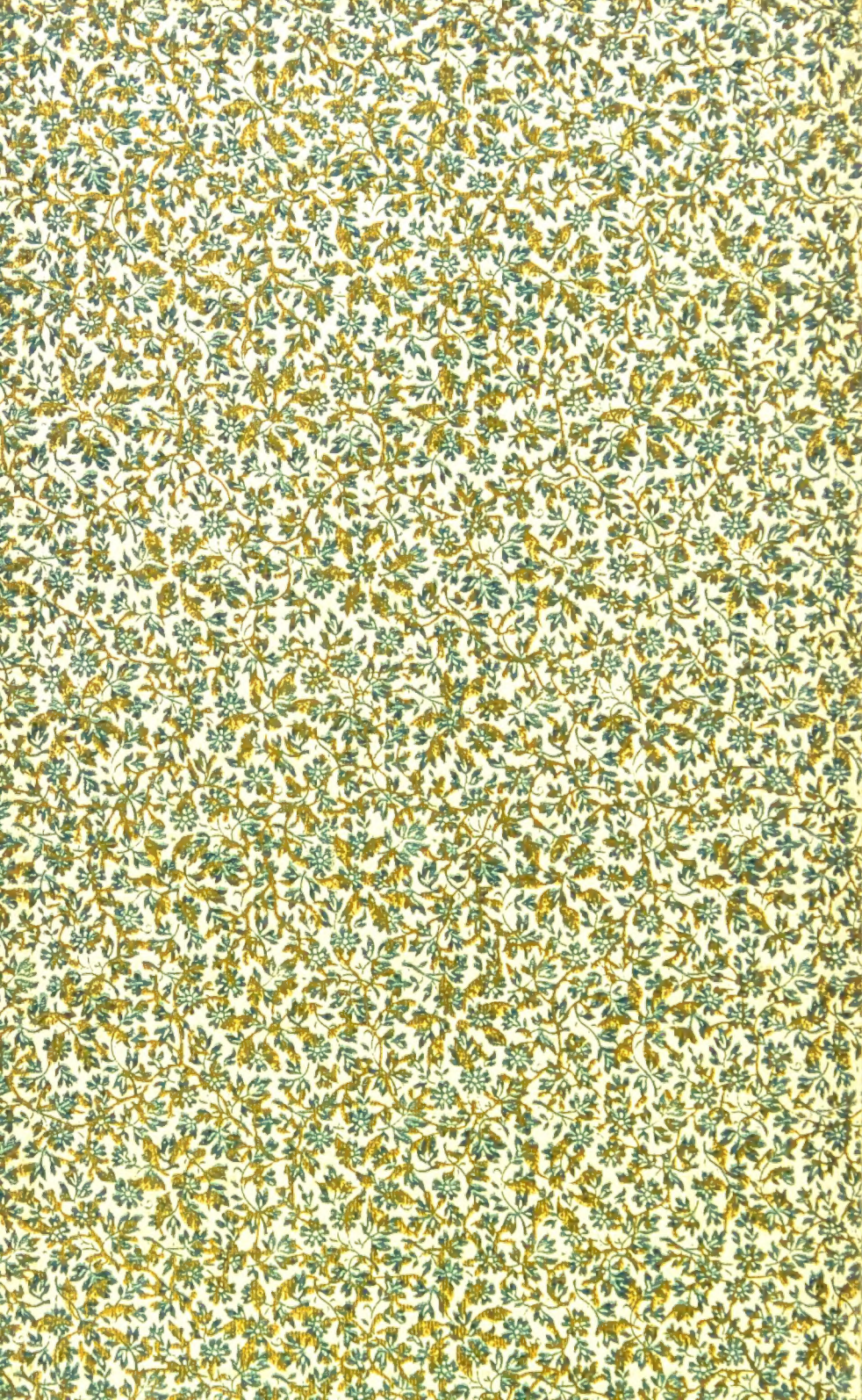






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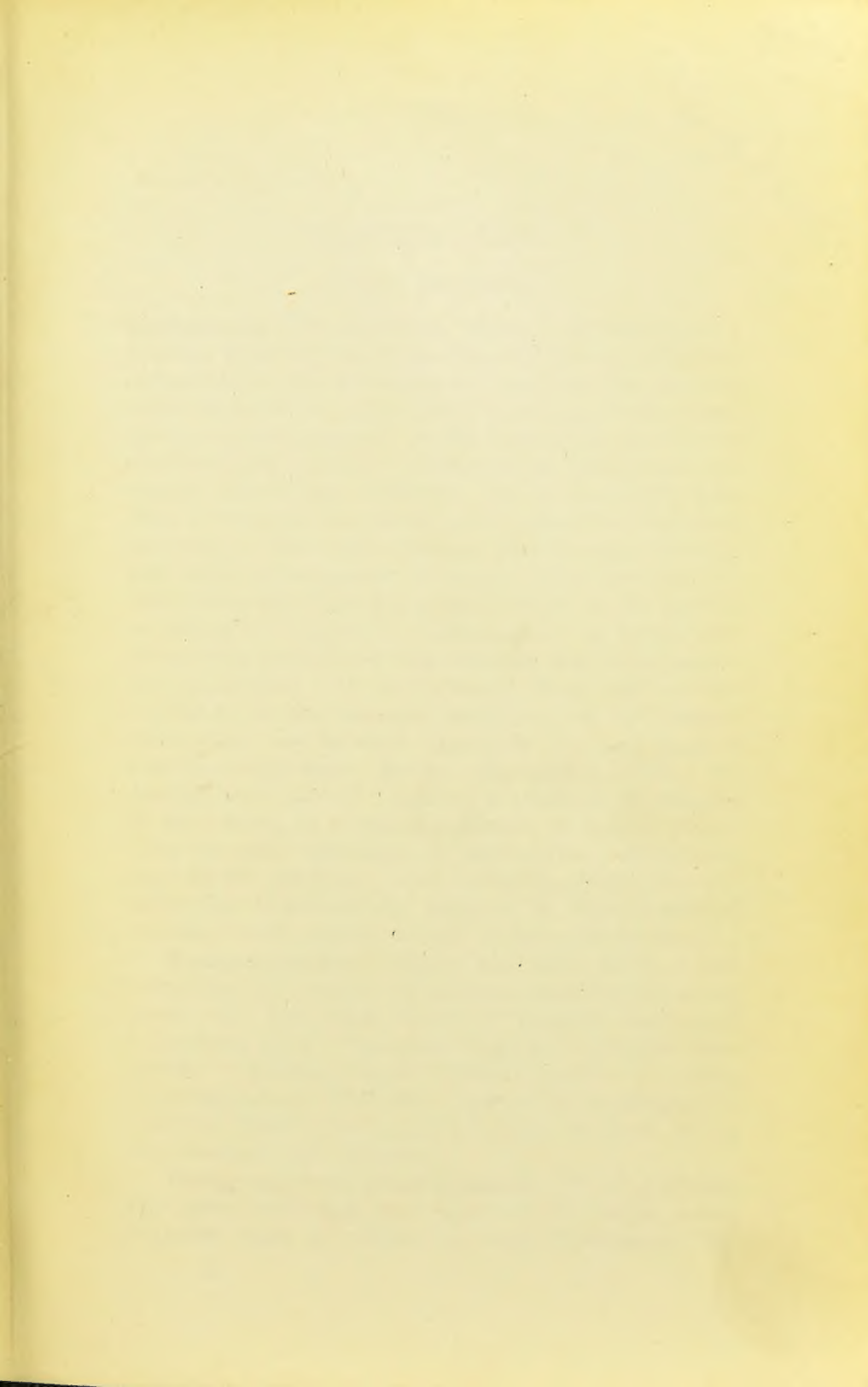
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CHAPTER XXIII.

SPRUE (PSILOSIS).

Definition.—By the term “sprue” is understood a peculiar and very dangerous form of chronic catarrhal inflammation of the whole or part of the mucous membrane of the alimentary canal, generally associated with suppression of the chologenic function of the liver and, probably, of that of the other glandular organs subserving digestion. It is frequently met with in tropical countries, particularly in European residents. The same disease may develop for the first time in temperate climates ; only, however, in individuals who have previously resided in the tropics or subtropics. Sprue is characterised by irregularly alternating periods of exacerbation and of comparative quiescence ; by an inflamed, bare, and eroded condition of the mucous membrane of the tongue and mouth ; by flatulent dyspepsia ; by pale, copious and generally loose, frothy, fermenting stools ; by wasting and anæmia ; and by a tendency to relapse. It may occur as a primary disease, or it may supervene on other affections of the bowels. It is very slow in its progress ; and, unless properly treated, tends to terminate in atrophy of the intestinal mucosa, which usually, sooner or later, proves fatal.

Nomenclature.—Sprue has been more or less recognised by writers on tropical medicine for many years. It has been called “tropical diarrhœa,” “diarrhœa alba,” “aphthæ tropicæ,” “Ceylon sore mouth,” “psilosis linguæ” (Thin), besides a variety of other names. The term “sprue” is an adaptation from the Dutch word “spruw” in use in Java, where the disease is very common.

Geographical distribution.—It is probable that sprue, although more common in certain warm countries than in others, is found throughout the

greater part of the tropical and many parts of the subtropical world. It is especially common in South China, Manila, Cochin China, Java, the Straits Settlements, Ceylon, India, tropical Africa, and the West Indies (Hillary). Apparently it is most prevalent in those countries in which high temperature is combined with a moist atmosphere. It is common, however, in certain subtropical countries, as North China and, occasionally, even in Japan; countries where, although the summer is hot and damp, the winter is dry and bracing.

Ætiology.—Prolonged residence in the endemic area is perhaps the most potent predisposing influence; cases, however, do occur in which the disease shows itself after a residence of one or two years only. Exhausting diseases, particularly those involving the alimentary canal, as dysentery, bill diarrhœa, morning diarrhœa, hæmorrhoids and fistula, are apt to terminate in sprue. Frequent childbearing, miscarriages, uterine hæmorrhages, exhausting discharges, and prolonged lactation also predispose to the disease; so may syphilis, courses of mercury or of iodide of potassium, bad food, bad water, anxiety, chills, and so forth—in fact, any depressing influence, particularly if it is combined with intestinal irritation. Malaria does not seem to be specially responsible. At one time *Anguillula stercoralis* (*Rhabdonema intestinale*), a parasite very common in the stools of cases of chronic intestinal flux, particularly in Cochin China (p. 595), was put forward as the cause of the chronic entero-colitis (for the most part sprue) of that country. Subsequent investigations have disproved this. Like the anguillula, *Amœba coli* may be present in the stools in these cases; but, similarly, it is in no way responsible for the disease. Neither has any bacterium which can be regarded with any degree of certainty as special to sprue been separated from the characteristic stools. In searching for the fundamental cause of this affection, the latency which the disease occasionally exhibits, and the fact that the first symptoms may

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not appear until months or even years have elapsed since the patient quitted the tropics, must be kept in view.

Symptoms. — *Variability.* — There is infinite variety in the combination and in the severity of the various symptoms of sprue, as well as in the rate of progress of the disease. In some instances it may be almost a subacute process running its course in a year or two ; in others, again, it may drag on intermittingly for ten or fifteen years. Much depends in this respect on the circumstances, the character, the care, the treatment, and the intelligence of the patient.

General symptoms in a typical case. — In an ordinary fully developed case the patient—who is generally dark or muddy in complexion and much emaciated—complains of three principal symptoms : soreness of the mouth, dyspeptic distension of the abdomen, looseness of the bowels ; the last being particularly urgent during the morning and earlier part of the forenoon. The patient may also complain of feeling physically weak, of loss of memory, and of inability to take exercise or to apply his mind. His friends will probably volunteer the information that his temper is irritable and unreasonable.

Mouth lesions. — If the mouth is examined, the soreness will be found to depend on a variety of lesions of the mucous membrane, which, though painful, seem to be of a very superficial character. These lesions vary considerably in intensity from day to day. During an exacerbation the tongue looks red and angry ; superficial erosions, patches of congestion, and perhaps minute vesicles appear on its surface, particularly about the edges and tip. Sometimes, from the folding consequent on swelling of the mucous membrane, the sides of the organ have the appearance of being fissured. The filiform papillæ cannot be made out, although here and there the fungiform papillæ may stand up, pink and swollen. If the patient be made to turn up the tip of the tongue, very likely red patches of superficial erosion, some-

times covered with an aphthous-looking pellicle, may be seen on either side of the frænum. On everting the lips, similar patches and erosions are visible ; and if the cheek be separated from the teeth the same may be seen on the buccal mucous membrane. Occasionally the palate is similarly affected ; very often in this situation the mucous follicles are enlarged, shotty, and prominent. The gullet and uvula may also be raw and sore.

In consequence of the irritation caused by these superficial and exceedingly sensitive lesions, the mouth tends to fill with a watery saliva which may dribble from the corners. If the patient attempts to take any sapid food, strong wine, or anything but the very blandest diet, the pain and burning in the mouth are intolerable ; so much so that, although perhaps ravenously hungry, he shirks eating. Not infrequently swallowing is accompanied and followed by a feeling of soreness and burning under the sternum ; suggesting that the gullet, like the tongue, is also in an irritated, raw, and tender condition. During exacerbations of the disease the condition of the mouth becomes greatly aggravated. Although during the temporary and occasional improvements it becomes much less painful, even then salt, spices, strong wines, and all kinds of sapid foods sting unpleasantly ; and the tongue, particularly along its centre, is seen to be bare and polished as if brushed over with a coating of varnish. At all times the tongue is abnormally clean and devoid of fur ; during the exacerbations it is swollen, but during the remissions, and when not inflamed, it is small, pointed, and, owing to the anæmic condition of the patient, yellowish like a piece of cartilage.

Dyspepsia.—Dyspepsia is usually much complained of, the feelings of weight, oppression, and gaseous distension after eating being sometimes excessive. Very likely the abdomen swells out like a drum, and unpleasant borborygmi roll through the bowel. Occasionally, though not often, there may be vomiting,







the vomiting sometimes being sudden and not always accompanied by feelings of nausea.

Diarrhœa.—The diarrhœa associated with sprue is of two kinds ; one chronic and habitual, the other more acute and, in the early stages, evanescent. The former is characterised by one or more daily discharges of a copious, pale, greyish, pasty, fermenting, mawkish, evil-smelling material. The latter is of a watery character, also pale and fermenting, the dejecta perhaps containing undigested food. In these latter circumstances the diarrhœa usually brings with it considerable relief to the dyspeptic distension, at all events for a time. When the mouth is inflamed the diarrhœa is usually more active. The stools during periods of quiescence may be confined to one or two in the early morning or forenoon ; during the later part of the day the patient is not disturbed. The stools, however, even in this quiescent phase, are always extraordinarily copious ; patients remark their phenomenal abundance. They are passed almost, or altogether, without pain. Not infrequently during exacerbations there may be a tender excoriated condition of the anus, and sometimes, in women, a similar condition of the vagina. Thin states that the stools in sprue are acid.

Types, history, course, and termination. Protopathic sprue.—There is a striking uniformity in the history of most cases of sprue. On inquiry, we shall probably learn that the patient has been suffering for months, or perhaps years, from irregularity of the bowels. This, we may be told, began soon after arrival in the tropics as a bilious morning diarrhœa. For a long time this morning diarrhœa went on, without interfering in any way with the general health. Later the mouth, now and again, became tender, little blisters or excoriations appearing for a day or two at a time about the tip of the tongue or inside the lips. These sore spots would come and go. Perhaps, from time to time, exacerbations of the mouth symptoms would be associated with a little

increase of diarrhœa. Gradually the stools lost their bilious character and became pale and frothy; dyspeptic symptoms, particularly distension after meals, now appeared. As time went on, these symptoms would recur more frequently and in a more pronounced form, following, almost inevitably, any little imprudence as regards food or exposure. The general condition now began to deteriorate; emaciation, languor, lassitude, and inability to get through the day's work satisfactorily, becoming more pronounced each summer until, finally, a condition of permanent invalidism was established. Should the disease continue to progress, the emaciation advances slowly but surely. Diarrhœa may be almost constant, and now no longer confined to the morning hours; the complexion becomes dark, sometimes very dark; the appetite, sometimes in abeyance, is more frequently ravenous; unusual indulgence in food being followed by increased discomfort, temporarily relieved by smart diarrhœa. Finally the patient is confined to the house, perhaps to bed. The feet become œdematous, and the integuments hang like an ill-fitting garment, the details of the bony anatomy showing distinctly through the dry, scurfy, earthy skin. Finally, the patient dies in a semi-choleraic attack; or from inanition; or from some intercurrent disease. Such is the history of an ordinary, mismanaged case of sprue.

Sprue secondary to dysentery.—When the disease has supervened on dysentery, we shall learn that the motions characteristic of the original dysenteric attack gradually changed in character; from being scanty, mucoid, bloody, and accompanied with pain and tenesmus, they became diarrhœic, pale, frothy, their discharge being followed by a feeling of relief rather than of pain. The mouth at the same time became sore, exhibiting the characters already described. Gradually a condition of confirmed sprue was established, which ultimately, unless properly treated, will almost certainly prove fatal.

Sprue secondary to acute entero-colitis.—Another

type of case commences as an acute entero-colitis with sudden and profuse colicky diarrhœa, vomiting perhaps, and a certain amount of fever. The acute symptoms do not subside completely, but gradually have the typical symptoms of sprue grafted on to those of an acute intestinal catarrh.

Incomplete sprue. (a) *Gastric cases.*—Occasionally we meet with cases of confirmed sprue in which, at first, the morbid process, judging from the existing clinical symptoms and subsequent history, is confined to a limited part of the alimentary canal. Thus we sometimes get sprue without diarrhœa, the principal symptoms being sore mouth, dyspeptic distension, pale, copious but solid stools, and wasting.

(b) *Intestinal cases.*—On the other hand, we may get cases in which the mouth is not ulcerated, and in which there is little or no distension or dyspepsia, but in which the stools are liquid, copious, pale, and frothy. Sometimes a patient may have suffered at an earlier period, or on a former occasion, from the first type of the disease, who, later, acquires the diarrhœic form; and *vice versâ*.

(c) *Sprue without diarrhœa.*—It sometimes happens that under treatment the sore mouth, the dyspepsia, and the diarrhœa completely subside; nevertheless the wasting continues, the stools remaining phenomenally copious—so much so that the patient may declare that more is passed than has been eaten. In this case wasting is progressive, and the patient gradually dies of inanition.

Intestinal atrophy consequent on sprue.—In certain instances, under treatment the symptoms proper to sprue subside; but the patient's digestive and assimilative faculties are permanently impaired. Slight irregularities either in the quality or the amount of food, chill, fatigue, depressing emotions, and other trifling causes suffice to bring on dyspepsia accompanied by flatulence and diarrhœa. These cases may linger for years; usually they improve during the summer in England, getting worse during the

winter and spring, or during cold damp weather. Ultimately they die from general atrophy, diarrhœa, or some intercurrent disease.

Morbid anatomy.—*Post mortem* the tissues in sprue are abnormally dry; fat is almost completely absent; the muscles and the thoracic and abdominal viscera are anæmic and wasted. With these exceptions and certain important changes in the alimentary tract, so far as known there are no special lesions which are invariably associated with this disease. According to Bertrand and Fontan, occasionally certain changes are present in the pancreas—namely, fatty or granular degeneration of the cells, with softening of isolated acini and slight inflammatory infiltration of the connective tissue. These, however, are not constant, any more than are certain other and similar changes occasionally found in the liver and kidneys.

Lesions of the alimentary tract.—The principal and characteristic lesions are found in the alimentary tract. The bowel is thinned to such an extent as to be almost diaphanous. The serous coat is generally healthy, the muscular coat atrophied. The submucosa in places has undergone hypertrophic fibrous changes; and the mucous membrane from mouth to anus, either in patches or universally, is superficially eroded and interstitially atrophied. The internal surface of the bowel is coated with a thick layer of dirty grey, tenacious mucus which conceals patches of congestion, of erosion, or even of ulceration, besides such evidences of similar antecedent disease as pigmented areas and thin scarred, cicatricial patches. The villi and glands are eroded and in many places completely destroyed. Here and there minute spherical indurations, about the size of a pin's head and surrounded by a dark pigmented or congested areola, can be felt in the mucous membrane. On cutting into these, they are found to be minute cyst-like dilatations of the follicles filled with a gummy muco-purulent material. Sections of the diseased

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bowel show under the microscope corresponding changes ; such as varying degrees of erosion or ulceration of the surface of the mucous membrane ; degenerations of villi, glands, and follicles ; the small mucous cysts referred to ; sometimes small abscesses ; and, also, infiltration by leucocytes of the basement membrane and submucous layer ; and, in the latter, fibro-cirrhotic changes. The mesenteric glands are generally large and pigmented, perhaps fibrotic. The erosion lesions are usually most marked towards the end of the ileum and in the colon ; but they may be present in greater or lesser degree universally, or in patches throughout the entire alimentary tract from mouth to anus.

Pathology.—In attempting an explanation of the phenomena of sprue, two features of the disease have to be considered—the catarrhal condition of the alimentary canal, and the absence of the normal colouring matter of the fæces. Possibly one of these is the consequence of the other ; possibly the two conditions are concurrent but independent consequences of the same cause. What that cause may be is quite unknown. Whether the first pathological step originates in physiological exhaustion of the digestive functions, brought about by tropical conditions abnormal to the European constitution ; or whether the disease depends upon a specific organism ; or whether there is a combination of these, has still to be settled. In view of the occurrence of morning diarrhœa of dark bilious stools as a frequent first step in the development of sprue, hyperactivity of the liver might be assumed to be a first step in the development of the disease, an activity which in time ends in exhaustion of the chologenic functions of the gland. It might be further suggested that, concurrently with this hepatic disturbance, there is a similar initial hyperactivity of all the other glands appertaining to digestion, a hyperactivity which also ends in a corresponding exhaustion. Chemical changes in the ingested food would then follow on

the establishment of these apeptic conditions, and ultimately, from the formation of acrid chemical bodies, lead to the chronic catarrhal changes found *post mortem*.

An analysis by Wynter Blyth of the stools in sprue resulted in ascertaining the presence of the ordinary elements of bile, notwithstanding their apparent absence so far as absence of colour would indicate. On the other hand, Bertrand and Fontan, in a similar analysis, failed completely to find bile acids. Until a decision is arrived at on this point it is useless to speculate further on the subject.

Micro-organisms, of course, abound in the fermenting stools of sprue; but hitherto no bacterium or protozoon which must be regarded as specific has been found in association with the disease.

Personally, I incline to regard sprue as an expression of exhaustion of the glandular structures subserving digestion, the result of over-stimulation by certain meteorological conditions which are found in tropical countries, and which are unsuited to the European constitution. The remarkable effect of physiological rest, as supplied by "the milk treatment," in curing sprue seems to support this hypothesis.

Diagnosis.—The condition of the tongue, the character of the stools, and the history are sufficiently distinctive, one would suppose, to render diagnosis an easy matter. Nevertheless, I have known of cases in which the disease has been diagnosed and treated as syphilis, the condition of the mouth being attributed to this disease, and the character of the stools and other symptoms being ignored.

Prognosis is good for recent cases, provided proper treatment is carried out. It is bad for patients over fifty, for long-standing cases, for careless and injudicious patients, and for those who cannot or will not take a pure milk diet.

Treatment.—*Importance of early and thorough treatment.*—If treatment be undertaken sufficiently

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early in sprue, and be thoroughly and intelligently carried out, it is generally marvellously successful. Should, however, it be undertaken at too late a period, when the glands and the absorbing surface of the alimentary canal have been hopelessly destroyed, do what we will, the case is sure to end fatally. In prescribing a treatment, therefore, the first thing for the physician to do is to get his patient thoroughly convinced of the deadly nature of his complaint; for, unless he receives the hearty and complete co-operation of his patient, the physician must not expect to cure a well-established case. To be successful, treatment must be thorough, sustained, and prolonged. All predisposing causes, as uterine or other discharges, syphilis, scurvy, and the like, must of course be dealt with and, so far as possible, removed.

The milk cure.—By far the most successful treatment is what is known as the “milk cure.” In carrying this out it is well to commence with a dose of some aperient—castor oil or pulvis rhei composita. Pending the action of the drug, all food, including milk, should be withheld. The patient should be sent to bed in order to economise strength and maintain an equable warm temperature of the skin. He should also be directed to clothe warmly, to encircle the abdomen with a broad flannel binder, to cover his arms and shoulders with a warm jacket, and to live in a warm room. When the purgative has acted the milk is begun. At first sixty ounces, at most, are allowed in the twenty-four hours, small quantities being given every hour or every two hours. When the patient is very weak the feeding must be continued during the night. The milk should not be drunk, but sipped with a teaspoon, or taken through a straw or fine glass tube, or from a child’s feeding-bottle. As a rule, on this regimen, in the course of two or three days, the patient’s condition is very much improved. The stools have increased in consistency — are solid perhaps, the distension of the

abdomen has subsided, dyspeptic symptoms have vanished, and the mouth is much less tender and less inflamed. The quantity of milk should now be increased at the rate of half a pint a day or every second day, until 100 ounces, or thereabouts, are taken in the twenty-four hours. It is well to keep at this quantity for ten days at least, when, everything going well, a gradual increase to six or seven pints may be sanctioned. Up to this point the patient should keep in bed ; but when he has reached this quantity he may get up and, if he feels strong enough and the weather is mild, go out of doors. *For six weeks, dating from the time the stools become solid and the mouth free from irritation, no other food or drink whatever should be permitted.* A raw egg, if it is found to agree, may now be added to the milk ; later, some artificial malted food ; next, small quantities of well-boiled arrowroot, rusks, pulled bread, thin bread (stale) and butter, or other digestible form of starchy food ; later still, chicken broth, a little fruit ; and, by-and-by, fish and chicken may be gradually introduced.

Importance of prompt treatment of threatened relapses.—Should, however, the slightest sign of dyspepsia or flatulence, especially of diarrhœa, or of sore mouth show itself, then the extra food must be discontinued immediately, a dose of compound rhubarb powder administered, and the patient be sent back to bed and placed once more at absolute rest and on a pure milk diet. In convalescents, no matter how long the acute symptoms have been in abeyance, this prompt recognition and treatment of threatened relapse should be rigorously observed. This is a rule of the utmost value and importance. Procrastination in treatment, under these conditions, is exceedingly dangerous. Promptitude in recognising and treating relapse not only saves time, but it may avert hopeless intestinal atrophy.

Symptoms persisting.—In commencing this treatment, if the patient after two or three days be

Barclay Wicks

found unable to digest and assimilate so much as three pints of milk in the twenty-four hours, the daily allowance must be reduced by half a pint a day until thirty ounces or thereabouts only are taken. If now the motions become solid, the quantity of milk must be gradually increased by five or ten ounces a day, so that in the course of a few weeks the full allowance—six or seven pints—is consumed.

How to meet inadequate assimilation.—It sometimes happens that the quantity of milk can be raised to seventy or eighty ounces per diem but no higher, further increase bringing on sore mouth, distension, and diarrhœa. In some of these cases the difficulty appears to depend not so much on digestion as on inability to absorb a large quantity of fluid. Occasionally, in such cases, one may succeed in getting the necessary amount of nutriment introduced by thickening the milk with condensed milk; or by slowly evaporating fresh cow's milk so as to reduce its bulk without diminishing the solids (Thin). The evaporation is best done in a vessel like a glue-pot, in which the milk is not boiled, but is surrounded by a jacket of boiling water; the milk during the process must be constantly stirred to prevent the formation of a scum. Or the milk diet may be supplemented by an adequate allowance of raw or underdone meat.

Other forms of giving milk.—Digestion is sometimes aided by peptonising the milk; or by mixing it with lime water or a little salt; or by aerating it in a soda-water syphon (*Goldsmith, Brit. Med. Journ., July 1st, 1893*). Koumiss sometimes agrees for a time when ordinary milk fails, and, if necessary, should be tried. Similarly, white wine whey is occasionally digested when milk is not; it is often of great service, especially when an alcoholic stimulant is indicated.

Treatment with meat juice and underdone meat.—Occasionally, symptoms persist or become aggravated under this system of treatment, and one is forced to conclude that milk does not suit the patient.

In such cases raw meat juice will often prove an efficient substitute. The juice of four or five pounds of fresh lean meat, and a little water to allay thirst, may be taken in small quantities at short intervals daily. After a time, when the stools are reduced in number and quantity, although perhaps not quite solid, scraped meat, or very much underdone meat, and by-and-by a little charred toast, a plain rusk or biscuit, and so forth, may be gradually added to the diet.

Meat and warm water diet.—Not infrequently, after the stools have become solid under a carefully regulated pure milk diet, it is found that any attempt to return to ordinary food, or to take anything beyond the most simple farinaceous dishes, is quickly followed by a recurrence of diarrhoea and the familiar flatulent dyspepsia. Such cases are sometimes successfully treated by a complete abandonment of milk, fish, and farinaceous stuffs for a time, and placing the patient on what is known as the “Salisbury cure.” This is a diet consisting only of meat and warm water. Commencing with smaller quantities, in time the allowance of meat is gradually raised to about three pounds *per diem*, taken at equidistant intervals in three or four meals. The meat must be of good quality, free from fat, coarse fibre, and gristle; it may be prepared as mince, or in the form of steak or chop, not too much cooked. Warm water, amounting in all to four pints in the twenty-four hours, is drunk before going to bed and on rising in the morning, and also about two hours before meals—never at meals. This course must be persisted in for six weeks, when ordinary food will be gradually attempted again.

Nutrient enemata or suppositories.—In all grave cases of sprue nutrient enemata or suppositories should be steadily administered every four or six hours. If tolerated they are most valuable aids to nutrition. It is well, when using them, to wash out the rectum once a day with cold water.

followed by a carefully selected &
more or less mixed diet

(Remember I have seen cases in which ^{the} after the
the most carefully carried out well, and
most pure diets a mixed diet proved
successful for a time. In such mixed diets
~~quantity~~ restriction in the amount of the
food ~~quantity~~ is almost as important an
element as quality.

The night ~~rest~~ to food very lightly.

These methods of treatment—milk and meat juice combined with warmth and rest—are the two most successful methods of treating sprue; should they fail, the chances of recovery are poor indeed.

When to send the patient to Europe.—When sprue develops in the tropics, if feasible, the patient should be sent to Europe as soon as possible. It is a mistake, however, to ship an invalid with his disease active on him, or if his end is manifestly not very far off. Diarrhœa should not be active when the patient is put on board ship. In every case provision, such as a cow or an abundant supply of sterilised milk, should be made for carrying on treatment during the voyage.

The clothing and general management.—Sprue patients returning to Europe ought to be especially careful in their clothing, and they ought to get out their warm clothes before the ship leaves the tropics. If their return is during the winter, they should arrange to remain in the South of Europe till at least late spring. Next to an unsuitable dietary, perhaps cold is the most prejudicial influence to which a sprue case can be exposed. A sprue patient ought never to feel cold; he ought always to wear thick flannels, thick stockings, and, when up and about, thick boots. In winter a chamois-leather waistcoat, provided with sleeves, is of great service. His rooms ought to be warm. He ought never to be fatigued; he ought to go to bed early and rise late; in fact, he ought to do everything in his power to avoid irritating the bowel, to guard against chill, physiological depression, and the necessity for copious eating. During the summer England is suitable enough as a residence; but during the cold winter and spring months some milder, drier, and more sunny climate must be sought out.

Drugs in sprue.—Experience soon teaches one to distrust medicines in sprue. Occasionally a gentle aperient or, if diarrhœa is watery and excessive, a few drops of laudanum are of service; but active drugging

of all sorts is, as a rule, in the highest degree prejudicial. If the mouth is very painful, cocaine—five grains to the ounce—brushed on before eating will deaden sensibility and, for a time at all events, relieve suffering. Constipation must be carefully avoided, and a simple enema used if necessary.

I think it right to state that two methods of drug treatment seem, in some cases, to have been followed by good results. One, advocated by Dr. Begg, lately of Hankow, consists in the administration of repeated doses of yellow santonin. He recommends one or two doses of castor oil to commence with, and, thereafter, five grains of santonin in a teaspoonful of olive oil once or twice a day for a week, diet being at the same time attended to. The other method has gained for an irregular practitioner in Shanghai some reputation; it consists in the repeated administration of purgatives alternately with or before the exhibition of large quantities—two teaspoonfuls at a time—of some form of carbonate of lime, believed to be powdered cuttlefish bone or powdered crabs' eyes. I have tried the santonin treatment without benefit to patients. I have also used cuttlefish bone: in one case with the result of permanently stopping the diarrhoea but not of arresting the progress of the disease. In this case, although diarrhoea was most effectively checked, yet massive solid stools continued to pass. After a few weeks the patient died from asthenia, notwithstanding a liberal diet which, apparently, was digested but not absorbed.

The sprue patient, if possible, ought not to return to the tropics. If compelled by circumstances to go back, he must exercise the utmost care with regard to his health, and avoid exposure, fatigue, cold bath, alcohol, and all excesses; take a minimum of, or avoid altogether, red meat; purge gently and go on absolute milk diet on the slightest sign of relapse.



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CHAPTER XXIV.

TROPICAL LIVER.

THE subject of liver disease is everywhere a difficult and complicated one. It is especially difficult in tropical countries ; for not only is the resident there liable to all the forms found in temperate climates, but he is exposed, in addition, to various potent predisposing and exciting causes of liver disease not present, or only present in a very mild degree, in more temperate latitudes. These additional causes of liver disease/inseparable from the tropics/are heat, malaria and, especially, dysentery. To these, too often, have to be added injudicious personal habits, a tendency to over-full and over-rich feeding, to over-stimulation by alcohol, and deficiency of muscular exercise.

The young European who finds himself in the tropics for the first time is surrounded very often by luxuries in the shape of food, wine, carriages, servants, luxuries to which he had not been accustomed perhaps in his home. At first the change, the excitement of novelty, and the high temperature act as stimulants to appetite, and the excessive loss of fluid by cutaneous transpiration creates a powerful thirst. Little wonder, therefore, that in such circumstances the youth, having the appetite and the opportunity of gratifying it, is apt to indulge in food and drink beyond safe physiological limits. He is made lazy by the heat ; he cannot exercise during the day, and when evening comes he prefers lounging on the verandah or hanging about the club bar to walking, or riding, or games. Very likely he sits up late at night, drinking and smoking, so that in the morning he is too sleepy to

ride out or take any other form of exercise. And so it comes about, what with a surcharge of aliment and alcohol, and the diminished activity of lung metabolism and excretion incident to high temperature and muscular inactivity, that a very large and unusual amount of physiological work is thrown on the liver. With this large amount of work there is a corresponding hyperæmia. This may be considered the first stage of tropical liver—hyperæmia from functional activity; up to this point it is a purely physiological condition.

Pushed a step farther this physiological hyperæmia passes into congestion with blood stasis and consequent diminished functional activity. Hyperæmia of a physiological character will be evidenced by increase of functional activity, and there will be a copious flow of bile, sometimes causing diarrhœa of a bilious character, particularly morning diarrhœa. But when the limits of physiological hyperæmia are passed, and congestion of a pathological character sets in, the consequent arrest of function will be evidenced by pale stools, perhaps diarrhœa of a pale, watery, frothy, fermenting character—in the last case the diarrhœa doubtless depending, in part at least, on fermentative processes set up in the contents of an alimentary canal no longer kept relatively aseptic by an adequate supply of healthy bile. Other symptoms of this condition are headache, furred tongue, scanty, high-coloured, loaded urine, a feeling of weight or fulness, or even of pain in the region of the liver, and, probably, enlargement of the percussion area and other physical signs of enlargement of that organ. One step farther and such a condition may pass into actual hepatitis attended with fever, smart pain in the liver, tenderness on percussion, and still more marked increase of the hepatic area.

A functionally very active hyperæmic organ is prone to inflammation, even on slight cause. In the case of the hyperæmic liver a common cause of inflammation is chill, such as may arise from a cold bath, a wetting,

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or from lying uncovered on a warm night in a current of air. The experienced resident knows this very well, and is at great pains to guard against such an occurrence. He very likely wears what is known as a cholera belt ; he sleeps, even on the warmest night, in flannel pyjamas, and with a thin blanket drawn over his abdomen ; during the day he wears a woollen singlet and very likely serge or thin tweed clothes. He does not sit down in damp clothes, and he has a great respect for a shower of rain. Besides chill, there are other causes which may convert the hyperæmia into congestion or inflammation ; a blow may operate in the same way, so may a surfeit of eating or drinking, so may exposure to the sun, so may an attack of malarial fever or of dysentery.

Treatment.—Nature sometimes effects a cure in these cases of hepatic congestion by establishing a smart diarrhœa. In the treatment of such cases we cannot do better than to imitate Nature, and even to supplement her efforts. A few doses of the sulphates, in the shape of some kind of bitter water or of Carlsbad salts, generally give prompt relief. But if the subject of such attacks does not profit by experience and mend his ways, very likely his liver, in time, will become chronically hyperæmic and extremely liable to intercurrent attacks of congestion of a character more or less acute. The subjects of this type of “liver” ought to be most careful in their habits. They must not lie abed too long ; they must not take cold baths ; they must not take cold drinks, nor expose themselves to cold in any form ; they must clothe warmly ; and they must eschew alcohol in every shape. Animal food they must partake of but sparingly ; and they should give the preference to fowl and fish over beef and mutton. Fruit and farinaceous food may be more freely partaken of, but over-eating in every form must be avoided. Exercise should be taken at least twice a day ; and, at least once in twenty-four hours, the exercise should be of such a character as to provoke perspiration. A gallop

on horseback, a smart game of tennis or rackets, are excellent hepatic stimulants. Occasionally, once a week or not so often, particularly when a sense of fulness or aching in the right side seems to indicate that all is not right with the liver, a dose of Carlsbad salts or bitter water, preceded perhaps by a few grains of calomel, may avert more serious trouble.

When hyperæmia becomes chronic, when the patient is continually suffering with "liver," he should leave the tropics for a time. Nothing relieves these cases of chronic congestion so quickly or so effectively as a visit to Carlsbad and a thorough course of the waters there, and of the dietetic restrictions imposed in the Carlsbad cure. This should be followed up by country life in England and the active pursuit of country sports; the usual precautions in the shape of warm clothing, avoidance of cold baths, chills, alcohol, and high living being scrupulously observed and a weekly saline purge taken.

A serviceable imitation of the natural Carlsbad water may be made by dissolving fifty-three grains of the powdered salt in a pint of boiling water.* This may be divided into three equal portions, which are to be sipped as hot as possible, at intervals of twenty minutes, on an empty stomach, first thing in the morning. While taking the solution gentle exercise, as moving about the room, should be indulged in. Breakfast must not be taken till an hour after the last dose. If the bowels are not gently acted on an increased quantity of the salts should be taken. During the course, which should be persevered in for three weeks, the diet must be carefully regulated; butter, fat, nuts, fruits, pastry, preserves, tinned foods, cheese, salads, wines, spirits, and beer are to be avoided. Too much meat must not be taken; a small meat meal once a day must suffice. The quantity of food, too, should be restricted, and clothing, exercise, and bathing be carefully attended to. Much

* A good substitute for Carlsbad salts consists of sod. sulph. 2 parts, sod. bicarb. 1 part, sod. chlorid. 1 part.

excellent advice on the subject of the Carlsbad treatment will be found in Surgeon-Major Young's book, "The Carlsbad Treatment for Tropical Ailments," published by Thacker, Spink & Co., Calcutta.

During severe attacks of acute congestion, or of severe hepatitis attended with fever and much local distress, the patient must be kept in bed and placed on a very low diet of thin broths, barley water, or rice water, or milk and water. He should be purged freely and often with salines. A large hot poultice, two feet or more in length by one foot in breadth, should be laid over the region of the liver; such a poultice to be effective should pass from the centre of the back to well over the epigastrium. Dry cupping sometimes gives marked relief. Muriate of ammonia has a certain reputation in these cases; it should be prescribed in twenty-grain doses every six or eight hours. I have often used it, but I cannot vouch for its virtues; it does no harm.

When such a hepatitis is associated with dysentery, should it resist these milder measures, thirty to sixty grains of ipecac. often give marked relief. This dose should be repeated every twelve or twenty-four hours for two or three times. When the hepatitis is associated with malarial fever, full doses of quinine, in addition to the purging and the other measures already mentioned, are indicated.

Whether hepatitis, unless associated with dysentery, ever passes on to suppuration is a moot point. Some say that it may; others resolutely deny that there is such a thing as "tropical abscess" unassociated with dysentery. This subject will be discussed in the following chapter. Malarial hepatitis has already been considered (p. 110).

CHAPTER XXV.

ABSCESS OF THE LIVER.

Definition.—A form of suppuration in the liver, occurring especially in warm climates, principally in male Europeans and in association with dysentery.

Geographical distribution.—Abscess of the liver, of the type known as tropical abscess, is, for the most part, a disease of warm climates. Usually a sequel, or, it may be, a concomitant of dysentery, it is rare or altogether absent in countries where dysentery is also rare or absent. Its geographical distribution, therefore, is in the main regulated by that of dysentery. It has to be noted, however, that liver abscess is not a sequel or concomitant of the dysentery of all countries and at all times. Thus it is rare as an indigenous disease in temperate climates, even in those temperate climates in which dysentery is at times common enough. Again, in tropical climates the dysentery and liver abscess curves do not everywhere and at all times maintain a constant and definite relation to each other; for, even in hot countries, the dysentery of some places is more apt to be followed by liver abscess than is the dysentery of other places; and, even as regards dysentery in the same place, some epidemics are more apt to be associated with liver abscess than others are. Nevertheless, on the whole, it may be laid down as a fairly general law that in the tropics and sub-tropics the liver abscess curve follows in the main the dysentery curve; and that the geographical range of liver abscess in these climates is the same as that of dysentery.

In Great Britain the liver abscesses met with occur most frequently in individuals who manifestly had contracted the disease in the tropics. As a disease of indigenous origin, notwithstanding the considerable amount of dysentery in lunatic



asylums and similar large public institutions in Great Britain, it is distinctly rare, though not so uncommon as is usually supposed. Of course this remark does not apply to those suppurations which are connected with ordinary pyæmia, with gall-stones, hydatids, pylephlebitis, and the like; it applies only to dysenteric and, possibly, if there be such a disease, to idiopathic abscess. In northern and central Europe it is much the same in this respect as in Britain. The disease is more frequent in southern Europe—in Italy, Greece, the Balkan peninsula, and south Russia; it is said to be particularly common in Roumania. In eastern Asia, even outside the tropical belt, it is far from rare: thus it is not uncommon in Japan, and it is a very notable feature in the morbidity of Shanghai and the coast of south China. In Africa it is common enough; indeed, some of the best modern studies of the disease have been made in Egypt and in the Algerian province of Oran. In the western hemisphere there is a corresponding distribution; fairly common in the tropics, it becomes progressively rarer as we proceed north and south. It is apparently less common in the West Indies than in India and the East generally. In the southern hemisphere, although the cooler parts of Australia seem to enjoy a practical immunity, the European in the Northern Territory and in the neighbouring island of New Caledonia is subject to this disease.

Ætiology.—*Relation to dysentery.*—There can be no question as to the existence of an intimate relationship between dysentery and liver abscess. Numerous and well-authenticated statistics, as well as everyday experience, attest this. In 3,680 dysentery autopsies made in various tropical countries, and collated by Woodward, 779 (21 per cent.) revealed abscesses of the liver. To quote recent Indian experience:—According to the Annual Report of the Sanitary Commissioner with the Government of India for 1894, out of 465 European soldiers who died from

dysentery in India during the period 1888-94, 161 (35 per cent.) had, in addition to dysenteric lesions, abscess of the liver. Conversely, in Egypt, Kartulis, in an experience of over 500 cases of liver abscess, elicited a history of dysentery in from 55 to 60 per cent.; Zancarol, also in Egypt, in 444 cases, elicited a similar history in 59 per cent.; and Edwards and Waterman, in 699 collated cases, elicited a like history in 72.1 per cent. During the period 1870-95, of 45 cases of liver abscess treated at the Seamen's Hospital Greenwich, and collated by Mr. Johnson Smith, *post-mortem* evidence, or a distinct history, of dysentery was obtained in 38 (84.4 per cent.).

These figures are conclusive as to the existence of an intimate relationship between dysentery and liver abscess. There is good reason, however, for believing that, while they represent the truth, they do not represent the whole truth, and that the association is even more frequent than they indicate. As has been pointed out by Dr. Neil Macleod and others, the occurrence of antecedent dysentery in cases of liver abscess is very often overlooked; for, without a *post-mortem* examination, it may be impossible to pronounce definitely on this point in every instance. It is also well known that extensive dysenteric ulceration may be present and yet give rise to no active subjective symptoms whatever. Moreover, it must be borne in mind that many patients suffering from liver abscess forget, or fail to mention, the occurrence of a previous dysenteric attack, and that they may mislead the physician by describing such an attack as "diarrhœa." Further, at *post-mortem* examinations, dysenteric lesions of a superficial and apparently trifling character are often either not sought for or they are overlooked. Consequently, although the evidence of antecedent dysentery may not be forthcoming in a proportion of cases of liver abscess, it must not be concluded from this that in these cases there had been no dysentery.

In a masterly paper (*Brit. Med. Journ.*,

~~October 26th, 1895~~) Macleod, after a very careful and critical analysis of certain figures bearing on this subject, concludes that dysentery is a factor in nearly every case of tropical liver abscess. In forty cases of the disease observed in Shanghai he had positive evidence of dysentery in all except one; and even in this case, as recovery ensued, there was no certainty that dysenteric lesions had not been present ~~in it also~~. Perhaps Macleod's conclusions are somewhat too sweeping; I confess, however, that they are, in the main, in harmony with my own experience. Doubtless they apply to liver abscess as met with in Shanghai and, probably, in many other places. It is just possible, however, that what holds good for one place may not hold good for all places, and that Bombay, for example, may differ in this respect from Shanghai. In the Sanitary Commissioner's Report, above referred to, it is stated that in 2 (3 per cent.) instances only, out of 74 cases of liver abscess occurring in the Bombay Presidency in the period 1888-94, were there dysenteric associations. It is difficult to believe that, did it always exist, so important and evident a circumstance as dysentery had been overlooked 72 times in 74 cases. It is equally difficult to believe that the liver abscess of Bombay is associated with dysentery in only 3 per cent. of cases, whilst, according to the same authority, in the whole of India it is certainly so associated in at least 30 per cent. of the total cases. Manifestly, the statistical aspect of this important question requires re-study in the light of more careful clinical and *post-mortem* observation.

Another important point, yet to be definitely settled, is the exact relationship in point of time of the dysentery to the liver abscess. In the great majority of cases the dysentery antedates the abscess. But many clinicians have held that in some instances the relationship is reversed; that in others the two diseases are from the commencement concurrent; whilst in others, again, hepatitis, presumably of a

kind which may eventuate in abscess, alternates with active dysenteric symptoms. If the abscess antedate the dysentery, then the dysentery cannot be the cause of the abscess. On these grounds some pathologists have regarded liver abscess and dysentery as but different expressions of one morbid condition; reacting to some extent on each other, but not directly related the one to the other as cause and effect. Here, again, the latency as regards symptoms of some dysenteries has to be discounted in attempting to settle the question.

Race and sex.—Besides this matter of its relationship to dysentery, there are several well-ascertained facts to be reckoned with before we can arrive at sound views on the subject of the ætiology of liver abscess.

(1) Though common in Europeans in the tropics, liver abscess is rare among the natives. Thus, in the native army of India the proportion of deaths from liver abscess to the total mortality in 1894 was only 0·6 per cent., whereas in the European army it was 7·4 per cent. Man for man, the relative liability of the European soldier and the native soldier was as 95·2 to 4·8.

(2) This disproportion is in spite of the fact that the native is more liable to dysentery than the European. Thus, in 1894, in the Indian army the admission rate among the native troops for dysentery was 43·8 per thousand, whereas in the European troops it was only 28·6; and in every hundred deaths in the native army 4·7 were from dysentery, against only 3·8 in the European army.*

(3) European women in the tropics, though quite as subject to dysentery as European men, rarely suffer from liver abscess; children hardly ever.

* Dr. Rees informs me that liver abscess is more common among the natives of Nigeria than these figures seem to show it to be among the natives of India. Among the negroes Dr. Rees informs me that the mortality in dysentery is much higher than among Europeans resident in Nigeria. Of those negroes attacked with dysentery who lived long enough many subsequently developed liver abscess.

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(4) The rarity of liver abscess in temperate climates.

Predisposing conditions.—The foregoing considerations seem to indicate that for the production of liver abscess at least two things are necessary—a predisposing cause, and an exciting cause. Dysentery is certainly not always and alone both the exciting and predisposing cause. Were this so, the native soldiers and the European women and children in India would suffer as frequently from liver abscess as do the European males there. Some additional factor evidently complicates the problem.

As liver abscess is developed principally in tropical climates and in European visitors there, and much more rarely in the native, it would seem that tropical conditions in those unaccustomed to them are in some way bound up with this predisposing element; and as liver abscess is rare in European women and children, it would seem that these conditions are in some way specially operative on European men. We have therefore grounds for concluding that, in addition to general tropical conditions, it must be the greater amount of exposure to which men, as compared with women and children, are subjected in the course of their business and amusements; or some other condition, especially that one which is relatively more common in men than in women and children—over-indulgence in stimulating food and alcoholic drinks—that constitutes this predisposing cause. Intemperate habits and exposure, doubtless, lead to a special liability in men to a hyperæmia and congestion of the liver tissue by which its resistive faculty to pathological influences is impaired. In these circumstances, pathological influences which in the healthier condition of the organ—such as we assume to exist more generally in natives and in European women and children—would have been successfully overcome, gain the upper hand and lead to suppurative disintegration of the organ. In support of this view we have the statement of Waring, that 65 per cent. of

liver abscesses observed by him were in alcoholics ; and it is also said, that when the native takes to European habits in the matter of eating and drinking, his liability to liver abscess is greatly and proportionately increased.

I conclude, therefore, that in the vast majority of instances the exciting cause of liver abscess is dysentery ; the predisposing cause hyperæmic, congestive, or degenerative conditions incidental to tropical life, supplemented by such things as exposure and unphysiological habits in eating and drinking.

Supplementary causes.—It is conceivable that in a highly predisposed liver exciting causes other than dysentery, such as a blow or sudden aggravation of chronic congestion by chill or excess, may suffice at times to determine suppuration. Liver abscess is most prone to develop at the commencement of the cold season. Further, one can conceive that in a hyperæmic liver struggling to resist dysenteric suppurative influences some third condition, such as the blow, chill, or surfeit referred to, may contribute to or determine the formation of abscess which, in their absence, might have been averted.

Briefly stated, the causes of liver abscess are, first, predisposing—hyperæmic and degenerative conditions of the liver ; second, exciting—dysentery, or dysentery combined with chill, dietetic excess, or traumatism.

Influence of age and length of residence.—Liver abscess may occur at any age after childhood, but is most common between twenty and forty. It is most prone to show itself during the earlier years of residence in the tropics (40 per cent. in the first three years), although the older resident is by no means exempt.

Influence of malaria.—Malaria, by causing frequent attacks of hepatic congestion and by lowering the general vitality, may have some predisposing influence ; but, as already pointed out, malarial hepatitis is essentially of a plastic and not of a suppurative nature. It is a common mistake to suppose that malaria causes the suppurative liver disease of the

tropics ; the two concur geographically, but are in no way ætiologically identical.

Organisms concerned.—The questions of the organisms concerned in liver abscess will be discussed in the section on pathology.

Symptoms.—There is great variety in the grouping of symptoms in liver abscess. The following is a common history.

The patient, after residing for some time in the tropics, during which he enjoyed good general health and lived freely, was attacked by dysentery. In due course he appeared to recover, and resumed work. Several weeks or months elapsed when, after a wetting, or some such incident, he began to feel out of sorts, to suffer from headache, foul tongue, want of appetite, irregularity of the bowels, disturbed nights, excessive and unaccountable languor, irritability of temper, and depression of spirits. About the same time he began to be conscious of a sense of weight and fulness in the right hypochondrium. Later, he became feverish, particularly towards evening, the oncoming of the febrile distress being sometimes preceded by a sense of chilliness. At times he had sharp stabbing pain in the right side in the region of the liver, perhaps a dry cough and, possibly, a gnawing, uncomfortable sensation or pain in the right shoulder. His friends observed that his face had become muddy and haggard. He was uneasy if he lay on his left side. The quotidian rise of temperature now became a regular feature, the thermometer every evening touching 102° —sometimes more, sometimes less—and sinking to near normal by morning. He now began to perspire profusely at night, and even during the day when he chanced to fall asleep. He had to change his sleeping clothes once or even twice every night on account of the drenching sweats. On examination it is found that the patient is somewhat emaciated ; his complexion thick and muddy ; his pulse 80 to 100 ; his tongue is furred and yellowish ; the palms of his hands and soles of his feet are cold and clammy. As

he lies on his back it is obvious, on inspection, that the epigastrium is too full for one so emaciated ; and it is seen that the breathing is shallow and mainly thoracic. The right rectus muscle is found to be rigid. Considerable discomfort, if not pain, is elicited by attempts at palpation and percussion over the right hypochondrium. The liver dulness extends an inch too high, and an inch or more beyond the costal border in the nipple line ; posteriorly, it rises to about the eighth rib in the line of the angle of the scapula. It is further observed that the line of dulness is arched along its upper border ; and that it is not materially altered by changes of position, unless it be on standing, when the lower margin descends markedly in the epigastrium. Percussion below the right costal border, on deep inspiration, gives rise to much uneasiness or even to acute pain. Very likely one or two tender spots can be discovered on firm pressure being made with the finger tips in some of the lower right intercostal spaces, or below the right costal margin. The spleen is not usually enlarged. Auscultation may detect pleuritic friction somewhere over the base of the right lung, or peritoneal friction over the liver itself. The urine, free from albumin, is scanty, high-coloured, and deposits ~~copious~~ urates on cooling.

As the case progresses emaciation increases ; hectic with drenching nocturnal sweats continues ; the liver dulness and pain may further increase ; or the general enlargement may somewhat subside, and percussion may reveal a pronounced local bulging, upwards or downwards. If the abscess which has now formed is not relieved by operation, after months of illness the patient may die worn out ; or the abscess, which has now attained enormous dimensions, may burst into the right lung or pleura, or elsewhere, and be discharged, and either recovery, or death from continued hectic and exhaustion or from some intercurrent complication, ensue.

The great variety in the urgency of symptoms.—
Although the foregoing is a fairly common history in

liver abscess, there are many instances in which the initial symptoms are much more urgent, and in which the disease progresses much more rapidly. In other instances subjective symptoms are almost entirely absent; or so subdued that the true nature of the case may be entirely misapprehended until the abscess bursts through the lung or bowel, or a fluctuating tumour appears in the neighbourhood of the liver; or, perhaps, not until after death, when the unsuspected abscess is discovered on the *post-mortem* table.

Fever.—In an acute sthenic case the initial inflammatory fever may run fairly high and persist for some time. Later, when it may be assumed that pus has formed, the fever becomes distinctly quotidian and intermittent in type, the morning temperatures being normal, or only slightly above normal, the evening rising to 101° or 102° , or a little over or under this. Sometimes evening temperatures of 103° , rarely of 104° , are registered. In the asthenic and insidious type, at first there may be short flashes of feverishness at more considerable intervals, to be followed later by a steadier fever of a hectic type, as in the suppurative stage of the sthenic cases. In either type there may be afebrile intervals of several days' duration; and in either there may also occur, concurrently with aggravations of the local conditions, spells of continued high temperature. Occasionally, though rarely, liver abscess may be unattended by fever of any description whatever.

Rigors.—In the classical descriptions of liver abscess the occurrence of violent rigor is generally mentioned as a notable sign of the formation of pus. Undoubtedly such a rigor does at times signalise this event; but it is by no means constant, and its absence is no guarantee that abscess has not formed. Generally the evening rise of temperature is preceded by a sense of chilliness, sometimes by a more marked rigor simulating, in the regularity of its recurrences and in its severity, the rigor of a quotidian malarial fever.

Sweating, particularly nocturnal sweating, and of a very profuse character, is an almost invariable accompaniment of liver abscess. The patient's clothes may be literally drenched with perspiration. Even during the day—particularly, as already mentioned, if he chance to fall asleep—the sweat may stand in beads upon the forehead and around the neck. This, like most of the other symptoms, may be temporarily absent, or, in a small proportion of cases, trifling.

The *complexion* is generally muddy, cachectic, and slightly icteric-looking; marked jaundice, however, is uncommon.

Wasting is generally decided and progressive.

Rheumatic-like pains and swelling of the hands and feet, such as occur in chronic septic affections, are sometimes to be noted. They usually disappear rapidly when the abscess bursts or is opened, and free drainage is established.

Pain of some description is rarely absent. In a few exceptional cases there is no pain; such a patient may declare that he does not know that he has a liver.

There are several types of pain—local and sympathetic—associated with liver abscess. Complaint is almost invariably made of a sense of fulness and of a sense of weight in the region of the liver, not infrequently referred to the infra-scapular region. Stabbing, stitch-like pain, increased by pressure, and especially by deep inspiration, coughing, and all sudden jarring movements, is very common, and probably indicates peri-hepatitis from proximity of the abscess to the surface of the organ. Percussion, or firm palpation, especially if practised during deep inspiration and below the ribs in front, generally causes smart pain and decided shrinking, the rectus muscle starting up as if to protect the subjacent inflamed parts. Pain on swallowing, at the moment the bolus of food traverses the lower end of the œsophagus was mentioned to me by a medical man,

himself the subject of hepatic abscess, as being a marked symptom in his own case. Pain on firm pressure with the finger tips in an intercostal space, and over a limited area, is a common and valuable localising sign. Among the sympathetic pains may be mentioned shooting pains radiating over the chest and down the right flank and hypochondrium.

Pain in the right shoulder.—This symptom is present in about one-sixth of the cases. It may be persistent, or it may intermit; it may radiate to the side of the neck, or to the region of the scapula, or down the arm; or it may be limited to the shoulder tip and clavicular region. In some instances it is of a dull, gnawing, aching character; in some it is more acute; and in some it may be represented by a burning sensation, as if the surface of the skin had been flayed by a blister. This symptom is a reflex transmitted from the hepatic terminals of the phrenic through the fourth cervical to the branches of the cervical and brachial plexuses.

Cough of a dry, hacking character, doubtless also a reflex from irritation of the diaphragm, or from an inflamed condition of lung or pleura over the seat of abscess, is not uncommon. When the abscess discharges through the lung, cough is sometimes very severe and may cause vomiting.

Respiration is generally shallow and proportionately rapid. This is partly symptomatic of the attendant fever; oftener it is owing to the fact that fuller inspiration is attended with stitch. Sometimes the breathing is entirely thoracic, the lower part of the chest seeming to be fixed—especially the right side—and the diaphragm almost motionless.

The *decubitus* is usually dorsal or right dorsal, the body being somewhat bent towards the right side and the right leg perhaps slightly drawn up. When the patient stands, a stoop to the right may be noticeable. Lying on the left side generally causes pain from dragging on adhesions, or discomfort from the pressure of the enlarged liver on the heart and

stomach. Occasionally the decubitus is indifferent, or even on the left side.

The *digestive organs* are usually disturbed and the tongue is coated. Vomiting may occur from time to time, arising either from pressure on the stomach by the swollen liver or as an expression of gastric catarrh; appetite, as a rule, is poor; flatulence may be troublesome; the bowels are confined or irregular, or there may be diarrhœa or dysentery. In the case of concurrent dysentery, it may be noted sometimes that the hepatic and dysenteric symptoms alternate in severity.

The *area of hepatic percussion dulness* is usually extended upwards and downwards, and sometimes horizontally. The extension may be general, especially in the earlier stages; later, careful outlining of the upper and lower boundaries may discover a limited and dome-like increase in one direction, most significant if upwards. The upper line of dulness is not, as a rule, horizontal, as in hydrothorax; almost invariably, on approaching the spine, it trends downwards more markedly than in hydrothorax or empyema. Variations in the extent of the dulness may take place from time to time, and sometimes very rapidly, depending on fluctuations—not in the size of the liver abscess, but on the varying and relative amounts of local and general hepatic congestion. One sometimes finds even a narrow hepatic dulness in the nipple line with a great increase in the axillary or scapular lines. In one case the lower border of the liver may be as low as the umbilicus; in another, especially in front, it may be well inside the costal margin. Diagnosis in the latter type of case is difficult, and depends rather on the nature of the fever, and on the history and general condition, than on local signs.

Splenic enlargement may be present even when there is no malarial complication. This is rare however and, in uncomplicated cases, is seldom great. I have seen splenic tumour closely simulated by abscess in the left lobe of the liver.

can be elicited ; it is seldom, however, as has already been insisted on, that careful inquiry fails to bring out some story of previous bowel disturbance more or less urgent. In a few instances liver abscess of tropical origin does not declare itself until the patient has been several years resident in a temperate climate and quite outside the endemic area.

The *incidence of the symptoms* is equally variable. Some cases commence with marked sthenic fever, much local pain, great tenderness and hepatic enlargement, signs of suppuration as rigor, hectic, and local bulging, rapidly supervening. Others, again, commence so insidiously that the patient can hardly say when he first began to feel ill ; perhaps there may be a history of slow deterioration of the general health during a year or longer before definite hepatic symptoms show themselves. The former type seems to be the more common in the young and robust newcomer to the tropics ; the latter, in the more or less cachectic and old resident. Between these extremes there is endless variety.

Duration of the disease.—Liver abscess may run its course in three weeks. Generally it is an affair of several months. Sometimes it may run on for a year or even longer ; particularly so if it burst through the lung and drainage be imperfect, in which event the cavity may keep on bursting and refilling at intervals for almost an indefinite period.

Terminations.—Apart from operative interference, liver abscess may terminate in various ways. It may end in spontaneous rupture leading to death or recovery. Death may also be brought about in other ways—by the severity of the local disease : by prolonged hectic and exhaustion ; by concurrent dysentery ; or by intercurrent disease as pneumonia, pulmonary abscess, empyema, peritonitis. Recovery may also ensue on the abscess becoming encysted, or, possibly, absorbed.

Rupture of the abscess.—Rendu in a series of 563 instances of abscess of the liver, compiled from various

sources, gives an interesting table showing the direction of rupture in 159 (28 per cent.) of the cases which opened spontaneously. This table may be summarised as follows :—

Rupture occurred into the	pericardium	in	1 case	0·13 per cent.
„	pleura	31	„	5·5 „
„	lung	59	„	10·5 „
„	peritoneum	39	„	6·9 „
„	colon	6	„	1 „
„	stomach and			
	duodenum	8	„	1·4 „
„	bile ducts	4	„	0·7 „
„	vena cava	3	„	0·5 „
„	kidney	2	„	0·3 „
„	lumbo-iliac			
	region	6	„	1 „

From this it will be seen that about 28 per cent. of liver abscesses rupture spontaneously, most generally into the lung or pleura.

Rupture into the lung.—If rupture takes place into the lung the abscess contents may be suddenly discharged, mouthful after mouthful of pus mixed with blood welling up or being coughed up. In a few instances, in such circumstances, death has occurred suddenly from the flooding of the lungs with pus. More commonly the discharge is effected gradually, a few drachms being brought up with each cough; in the aggregate this discharge may amount perhaps to five or ten ounces in the twenty-four hours. In favourable cases the daily amount expectorated gradually diminishes until all discharge ceases and the patient recovers. Frequently, however, a deceptive arrest of discharge and cessation of cough are followed by a rise of temperature, which had become normal on the occurrence of rupture. With this there may be a reappearance of the night sweats. In a few days cough and expectoration return as before and fever once more subsides. This process of alternate emptying and refilling of the abscess cavity may recur many times before recovery finally takes place. In some cases it continues for months, and

may finally wear out the patient. Abscess may form in the lung; or a sudden and fatal hæmoptysis be brought about by ulceration opening some large pulmonary vessel. In some, expectoration never altogether ceases; if accompanied by fever this persistency indicates imperfect drainage, or, possibly, the presence of a second and unruptured abscess.

Characters of the expectorated liver pus.—The appearance of expectorated liver pus is almost pathognomonic. In colour it is chocolate brown; in consistence it is viscid and jelly-like. It may be streaked with blood; sometimes the expectoration may be almost entirely pure blood. Not infrequently these hæmorrhagic cases are regarded and treated as examples of ordinary hæmoptysis. Presumably, in the majority of instances, this blood comes from the wall of an abscess jarred and torn by the succussion of the harassing cough. Under the microscope expectorated liver pus exhibits the appearance to be presently described (p. 392).

Rupture into the pleura leads to sudden development of evidences of pleural effusion, which, unless relieved by drainage, may, in its turn, give rise to all the signs of empyema, and terminate in death, or in rupture through the lung or chest wall.

Rupture into the stomach is generally signalled by vomiting of the characteristic pus and, at all events temporarily, by cessation of local symptoms and fever.

Rupture into the bowel may cause diarrhœa, the pus more or less altered appearing in the stool. This is an occurrence that is frequently overlooked.

Rupture into pericardium, into peritoneum, or into a blood-vessel is almost necessarily and rapidly fatal.

Rupture through the skin is said to be the most favourable, though a rare, termination of liver abscess.

Mortality.—Rouis (203 cases), in Algiers, observed a mortality of 80 per cent.; Castro (125 cases), in Egypt, a mortality of 72·5 per cent. or,

excluding cases operated on, of 76 per cent. In the Indian army, during the period 1891-94 (prior to which abscess of the liver, in the statistical returns, is not separated from hepatitis), and, presumably, including cases operated on, the mortality was 57·7 per cent.

Causes of death.—In Rouis's 162 fatal cases the causes of death are stated as follows:—Severity of the local disease, or through the associated dysentery, 125; bursting of the abscess into the peritoneal cavity, 12; into the pleura, 11; gangrene of abscess wall, 3; peritonitis, 3; pneumonia from effusion of liver pus into the lung, 3; rupture of adhesions, 2; pneumonia, 2; rupture into the pericardium, 1.

Morbid anatomy.—It may be inferred from the symptoms that in the early stages of suppurative hepatitis there is general congestion and enlargement of the liver; in some instances this condition may be more or less confined to one lobe or even part of a lobe. Later, as we know more especially from observations in cases that have died from the attendant dysentery, one or more greyish, ill-defined, anæmic, circular patches, half to one inch or thereabouts in diameter, in which the lobular structure of the gland cannot be made out, are formed. These grey spots are very evident on section of the organ. A drop or two of a reddish, gummy pus may be expressed from the necrotic patches—for such they are. Still later, the centres of the patches liquefy and distinct but ragged abscess cavities are formed. An abscess thus commenced extends partly by molecular breaking down, partly by more massive necrosis of portions of its wall; partly by the formation of additional foci of softening in the neighbourhood and subsequent breaking down of the intervening septa. The walls of such an abscess have a ragged and rotten appearance. Spherical on the whole, there may be one or more diverticula extending from the main cavity; or contiguous abscesses may break into each other and communicate by a sinus. Occasionally a

thickened blood-vessel is met with stretching across the cavity. Though the pus and detritus lying on the abscess wall are viscid and adhesive, there is no notable exudation of lymph either lining the cavity, or in the still living liver tissue beyond. There is a peripheral zone of hyperæmia ; beyond this zone the gland may appear normal or simply congested

Number and size of abscesses.—Liver abscess may be single or multiple. If multiple, there may be two, three, or many abscesses. Zancarol's statistics, applying to 562 cases, give the proportion of single to multiple abscess cases as three of the former to two of the latter.

When single the abscess sometimes attains a great size. Frequently it is as large as a cocoanut or even larger ; it has happened that the entire liver, with the exception of a narrow zone of hepatic tissue, has been converted into a huge abscess sac. When multiple the individual abscesses are generally smaller, ranging in size from a filbert to an orange.

As might be expected from considerations of the relative size of the parts, abscess is more common in the right than in the left and smaller lobes. Roux gives the proportions in 639 cases as 70·85 per cent. right lobe, 3 per cent. left lobe, and 0·3 per cent. lobus spigelii.

Adhesions to surrounding organs are frequently, though not invariably, formed as the abscess approaches the surface of the liver. In this way the danger of intra-peritoneal extravasation is usually averted.

Pulmonary inflammation and abscess from escape of liver pus into the lungs are sometimes discovered *post-mortem*. Generally the pulmonary abscess communicates with the mother abscess in the liver by a small opening in the diaphragm.

Liver pus.—As already mentioned, the naked-eye appearance of liver pus is peculiar. When newly evacuated it is usually chocolate-coloured and streaked with, or mixed with larger or smaller clots or streaks



of blood, and here and there with streaks of a clear mucoid material. It is so thick and viscid that it will hardly soak into the dressings, but lies on the surface of the gauze like treacle on bread; spreading out between the skin and the dressing, and finding its way past the edge of the latter rather than penetrating it. When quite fresh, here and there little islands of yellowish what may be described as laudable pus may be made out in the brown mass. Sometimes it contains considerable pieces of necrotic tissue. Occasionally, from admixture of bile, the abscess contents are green-tinged. Liver purulage has always a peculiar mawkish odour; it is rarely offensive, unless the abscess lie near the colon, in which case it may have a faecal odour. Under the microscope many blood corpuscles are discoverable, besides much broken-down liver tissue, large granular pigmented spherical cells, débris, oil globules, hæmatoidin crystals and, occasionally, Charcot Leyden crystals, amœbæ, leucocytes, and the ordinary pyogenic bacteria.

Amœba coli and pyogenic organisms.—According to my experience of tropical abscess of the liver seen in England, *Amœba coli* can be detected in considerably over half the cases. This agrees with Kartulis's experience in Egypt, and that of others elsewhere. I have observed in a good many instances in which I have failed to detect the amœba in the aspirated liver pus, or in the pus which escaped at the time of operation, that the parasite appeared, often in great profusion, four or five days later in the discharge from the drainage-tube. I have seen them in these circumstances in strings of eight or ten, the string-like arrangement suggesting that they had developed in some tube, such as a blood-vessel. The amœbæ persist in the discharge until the abscess has healed. It is justifiable to infer from the absence of amœbæ in the pus constituting what might be called the body of the abscess, and their appearance in the pus coming from the walls of the abscess a few days later, that the habitat of the parasite is not so much the pus occupying

the general abscess cavity as that immediately in contact with the wall and the breaking-down tissues themselves. This is an inference entirely in harmony with Councilman and Lafleur's demonstration, confirmed by Marshall (*Brit. Med. Journ.*, June 10, 1899), of the parasite in the still living tissues around the abscess. In my experience the presence of the amœba does not affect prognosis unfavourably; Lafleur ("System of Medicine," Allbutt, vol. ix., 1897) says it does; I cannot agree with him, as I have many times seen amœbic liver abscess cases recover completely and rapidly after operation.

Other protozoa have been found in liver pus. Thus both Grimm and Berndt have found numerous active flagella therein. Some time ago, in the expectorated pus from a liver abscess discharging through the right lung, I found a ciliated infusorium resembling *Balantidium coli*.

In the pus of a large proportion of liver abscesses, both microscopic examination and culture may fail to detect the usual pyogenic micro-organisms. To harmonise this well-established fact with modern views on the cause and nature of the suppurative process, it has been suggested that, though in these sterile abscesses micro-organisms had originally been present, they had subsequently died out. This view receives a measure of support from the fact that in a proportion of instances there is no difficulty in demonstrating in the pus the ordinary pyogenic bacteria and, sometimes, the *Bacterium coli commune*. It by no means follows from this, however, that bacteria are a necessary factor in the production of all liver abscesses.

Encystment.—In rare instances the pus of liver abscess, instead of possessing the chocolate colour and viscid consistency described above, is yellow and creamy. This is particularly the case when the abscess becomes encysted—an occasional event. The walls of these encysted abscesses are thick, smooth, resistant, and fibrous. In time their contents become cheesy, and ultimately cretified; in the





latter event the cyst shrivels up and contracts to a small size.

Pathology.—The pathology of liver abscess has been a fruitful source of speculation and controversy. Much confusion has crept into the question from attempts to separate, ætiologically and pathologically, multiple from single liver abscess. The former is often called “pyæmic abscess” or “dysenteric abscess,” and has been set down as being the peculiar sequel of dysentery; the latter has been called and considered the “tropical abscess” *par excellence*, and regarded as idiopathic and entirely unconnected with dysentery.

As already pointed out, a careful examination of cases and statistics shows that both forms of abscess, single and multiple alike, are, in the vast majority of instances, clearly associated with dysentery. The dysenteric association, therefore, will not hold as a basis of classification and distinction. In their respective clinical histories, in their symptoms, in the characters of their walls and contents, in the frequent presence of *Amœba coli*, single and multiple abscesses are practically identical. The only difference between them is a numerical one; also a circumstance quite inadequate to base a doctrine of specific distinction on.

The view which I incline to hold on this subject has already been partly indicated in the section on ætiology. There are two factors which are principally concerned in the production of liver abscess: (1) the predisposing—weakening of the resistive faculty of the liver by chronic congestion or tissue degeneration, and, perhaps, other subtle changes brought about by a combination of climatic, dietetic, and other tropical conditions; (2) the exciting—some micro-organism, streptococcus, staphylococcus, *Bacterium coli commune*, amœba, or other parasite which, coming from the ulcerated dysenteric colon, or by way of the portal circulation (Marshall has recently demonstrated *Amœba coli* in a thrombus in

a branch of this vessel), gains access to the liver and proliferates in the weakened tissues. In at least 90 per cent. of cases the pyogenic micro-organism is derived from dysenteric processes in the colon. Whether the resulting abscess be single or multiple is more or less a matter of accident. If the weakened liver is efficiently inoculated at one point only, there is only one abscess; if at many points, then there is multiple abscess. This is virtually, in a sense, Budd's theory expressed in modern terms.

An apparently weighty objection to this view is sometimes urged. Why, it is asked, if liver abscess be the result of septic absorption from a dysenteric ulcer, is it not a common sequel of typhoidal or of tuberculous ulceration in the tropics? Macleod has met this objection very ingeniously and, I believe, to a certain extent, correctly. He points to the fact that typhoidal and tuberculous ulcerations are surface lesions unattended with abscess formation in the wall of the bowel. In their case there is free escape of the products and germs of ulceration; whereas in dysenteric lesions, in addition to the superficial ulceration, there is often what is really abscess formation with burrowing and retention of pus below the mucous membrane, and therefore great liability to entrance of micro-organisms into the radicles of the portal vein. Liver abscess, therefore, according to this view, is a pyæmic process. Often, however, it must be confessed, the dysentery preceding liver abscess appears, judging from the symptoms, to be of the catarrhal rather than of a more severe type.

To what extent *Amaba coli* is concerned as an occasional, or even common, cause of liver abscess it is as yet impossible to state. But if we watch the movements of this animal on the warm stage; and if we reflect that it lives and wanders about in the same very active way among the structures forming the walls of the liver abscess, and even in what are comparatively sound tissues, preceding, as it were, the suppuration; and consider that it lives at the

expense of these tissues, it is hard to resist the conclusion that the amœba operates as a disintegrating and irritating agency. Kartulis suggests that it may act merely as a carrier of pus-forming bacteria. Others maintain that it is a harmless epiphenomenon, incapable in any way of inducing pus-formation.

Calmette, in view of the frequency with which he and others have found liver abscess to be sterile, suggests that the exciting agency is of a chemical nature, some irritating liquefying body derived from the decomposition processes going on on the surface of the dysenteric ulcer.

Diagnosis.—Of all the grave tropical diseases none is so frequently overlooked as abscess of the liver. Acute sthenic cases are readily enough recognised; not so the insidious asthenic cases. The novice in tropical practice is some time in realising that grave disease of so important an organ as the liver may, for a long time, be unattended with urgent symptoms, whether local or constitutional, or both.

The most common mistakes in diagnosis are : (1) Failure to recognise the presence of disease of any description, even when an enormous abscess may occupy the liver. (2) Misinterpretation of the significance and nature of a basic pneumonia—a condition so often accompanying suppurative hepatitis. (3) Attributing the fever symptomatic of liver abscess to malaria. (4) Mistaking other diseases for abscess of the liver and *vice versa*—for example, hepatitis of a non-suppurative nature, such as that attending malarial attacks; suppurative hepatitis before the formation of abscess; syphilitic disease of the liver—softening gummata, which are often attended with fever of a hectic type; pyelephlebitis; suppurating hydatid; gall-stone and inflammation of the gall-bladder; subphrenic abscess; abscess of the abdominal or thoracic wall; pleurisy; encysted empyema; pyelitis of the right kidney; pernicious anæmia; leucocythæmia; scurvy and similar blood diseases associated with enlargement of the liver;

ulcerative endocarditis. Any of these may be attended with fever of a hectic type, increased area of hepatic percussion dulness, and pain in or about the liver.

Many times a correct diagnosis can be arrived at only by repeated and careful study of the case in all its aspects. Golden rules in tropical practice are to think of hepatic abscess in all cases of progressive deterioration of health ; and to suspect liver abscess in all obscure abdominal cases associated with evening rise of temperature, and this particularly if there be enlargement or pain in the liver and a history of dysentery — not necessarily recent dysentery. If doubt exists, there should be no hesitation in having early recourse to the aspirator to clear up diagnosis.

As bearing on prognosis, apart from the risk from sudden rupture in some untoward direction, to overlook abscess of the liver is a much graver error than to mistake some other disease for liver abscess ; for the chances of recovery from operation are proportionately prejudiced by every day's delay.

Low pneumonia of the right base in a tropical patient should always be regarded with suspicion ; in most instances it means abscess of the subjacent liver.

Perhaps the most common error is to regard the hectic of liver abscess as attributable to malaria. The regularity with which the daily fever recurs, the daily chilliness or even rigor coming on about the same hour, the profuse sweating, and other circumstances so compatible with a diagnosis of malaria, all contribute to this mistake. So common is the error that Osler says he hardly ever meets with a case of liver abscess which has not been drenched with quinine. My experience is the same. I have seen medical men make this mistake, not only in their patients, but in their own persons. If carefully considered, there are several circumstances which should obviate so serious an error. (1) No uncomplicated ague resists quinine in full doses. (2) In malaria,

if the liver be enlarged the spleen is still more so ; the reverse is the case in liver abscess. (3) The plasmodium cannot be found in the blood in non-malarial hepatitis. (4) In liver abscess the fever is almost invariably an evening one ; in malaria it most frequently comes on earlier in the day. (5) Quotidian periodicity, contrary to what is the case with tertian or quartan periodicity, is by no means pathognomonic of, or peculiar to, malaria. (6) The almost invariable history of antecedent dysentery, or, at least, of bowel complaint, in liver abscess.

To mistake other forms of suppuration for liver abscess is of no great moment, because in many of the suppurative diseases just enumerated the treatment is the same as for liver abscess, and no bad result need be looked for if diagnosis is not quite accurate. A more serious error, however, is to overlook the presence of leucocythæmia, pernicious anæmia, or scurvy, and to proceed to aspirate an enlarged liver on the supposition that the symptoms arise from abscess. Fatal intraperitoneal hæmorrhage from the puncture has been known to ensue in such circumstances. If any doubt exists on this point, a microscopic examination of the blood should be made before proceeding to explore.

A point to note in exploring is, that when the instrument enters the liver, an up-and-down pendulum-like movement will be communicated to the outer extremity of the needle in harmony with the rising and falling of the organ in respiration. If the needle does not exhibit this movement, its point may be in an abscess cavity, but this abscess is not in the liver.

Treatment.—Hepatitis which has not proceeded to abscess formation should be treated, in the absence of dysentery, with free purging by the sulphates, massive hot poultices, low diet, and rest in bed. If there be much pain, relief may be afforded by either wet or dry cupping over the liver, or by leeches around the anus. Ammonium chloride, in twenty-grain doses three times a day, is usually prescribed. If

dysentery be present, the case should be treated with full doses of ipecacuanha, repeated once or twice a day for two or three days, or by a cautious use of the purgative sulphates, and by poultices, rest, and low diet.

When the occurrence of rigor, or the development of hectic, or the appearance of local bulging, or the persistency of the fever and of the local symptoms, gives grounds for suspecting that abscess has formed, active medication must be suspended, a somewhat improved dietary prescribed, and measures taken without unnecessary delay to locate by means of the aspirator the position of the pus.

When he proceeds to use the aspirator, the surgeon must be prepared to open and drain the abscess if pus be discovered ; once diagnosis is established, nothing is gained by delay. By proceeding to open the abscess at once the shock of a double operation is avoided, and only one administration of the anæsthetic is required.

To facilitate aspiration, as well as the subsequent operation if such should be found to be necessary, the patient ought invariably to be placed under an anæsthetic. Unless in very special and exceptional circumstances, it is a mistake to attempt exploration without this, for the surgeon ought to proceed with deliberation and to feel himself at liberty to make as many punctures as he may think necessary. A medium- or full-sized aspirator needle should be used, as owing to the nature of the pus it may not flow through a cannula of small bore.

If there are localising signs, such as a tender spot, a fixed pain, localised bulging, localised pneumonic crepitus, pleuritic or peritoneal friction, these should be taken as indicating, with some probability, the seat of the abscess and the most promising spot for the exploratory puncture. If none of these localising signs are present, then, considering the fact that the majority of liver abscesses are situated in the upper and back part of the right lobe, the needle should, in the first instance, be inserted in the axillary line in the eighth or ninth interspace, about an inch







or an inch and a half from the costal margin and well below the limit of the pleura. The instrument should be carried in a direction inwards and slightly upwards and backwards and, if found necessary, to its full extent. If pus be not struck the needle must be slowly withdrawn, a good vacuum being maintained the while in case the abscess has been transfixed and the point of the needle lodged in the sound tissue beyond. No pus appearing in the aspirator the remainder of the dull hepatic area must be systematically explored, both in front and behind, regard being had for the lung and pleura on the one hand, and for the gall-bladder, large vessels, and intestine on the other. The peculiar colour—often like dirty brown thick blood—of liver pus must not be allowed to deceive the operator into thinking that he has failed to strike the abscess.

At least six punctures should be made before the attempt to find pus is abandoned. Provided there is complete absence of breath sounds, of vocal fremitus and resonance over the lower part of the right lung, and pus has not been reached from lower down, then the pleura or lung may be disregarded and puncture made anywhere below the line of the nipple and angle of the scapula, or wherever the physical signs suggest.

The surgeon should be encouraged to make early use of the aspirator by the fact that its employment, even where no pus is discovered, is not infrequently followed by rapid improvement in all the symptoms; many such cases are on record. Hepatic phlebotomy, as Dr. George Harley designated the removal from the liver of a few ounces of blood by the aspirator needle, is a measure of proved value in hepatitis. With due care, risk from hæmorrhage is small; it is very small indeed in comparison with the risk of allowing a hepatic abscess to remain undiscovered and unopened.

It is hardly necessary to add that strict aseptic precautions, in the way of purifying the patient's skin, the surgeon's hands, and all instruments, must be carefully observed.

Operations for abscess of the liver.—The following is the operation usually practised by English surgeons. It is substantially that described by Mr. Godlee in the *British Medical Journal* of January 11th, 1890, to which the reader is referred for many valuable details and practical hints.

If pus is struck below the costal border, the aspirator needle being left *in situ* as a guide, the abdominal wall is incised down to the peritoneum. A three-inch incision will give plenty of room. If firm adhesions be discovered, a sinus forceps is at once run along the needle, and pushed through the intervening liver tissue and into the abscess. The aspirator cannula is now removed, and the blades of the forceps are opened sufficiently, as it is being withdrawn, to make a wound in the liver big enough to admit the fore-finger, which must now be inserted and moved about so as to enlarge the wound and to gain some idea as to the size and direction of the cavity of the abscess. A rubber drainage-tube, about as large as the finger, and provided with a flange, is cut to a suitable length, and carried by means of the forceps to the back of the abscess. The abscess is then allowed to empty itself. When pus no longer flows freely, a massive antiseptic dressing is applied and firmly secured by a broad binder or many-tailed bandage.

If after division of the abdominal wall no reliable adhesions be discovered between this and the liver, the capsule of the latter must be securely attached to the former by a double circle of stitches. The abscess is then to be opened, as above described, with sinus forceps. After stitching, some surgeons prefer before opening the abscess, first to stuff the wound in the abdominal wall with iodoform gauze, and to wait for a day or two for adhesions to form. Others stuff the wound with gauze without previous stitching.

Should the abscess be struck through an intercostal space, and if the latter be not deemed sufficiently wide to admit of manipulation and free drainage, a couple of inches of rib had better be



excised. The diaphragm may then be stitched to the thoracic wall or, better, to the skin as well, when the abscess may be opened with forceps. To stitch the capsule of the liver to the diaphragm is a somewhat difficult proceeding; but if there are no reliable adhesions it had better be attempted, especially if the opening is to be made through a part of the liver covered by peritoneum. If, by any chance, the pleura is opened during the operation pneumothorax will result, an unfortunate, but not necessarily a serious, contingency. In this case the hole in the pleura must be carefully stitched in such a way that the pleural cavity is completely cut off before the diaphragm is divided and the abscess opened. Pus must not on any account be allowed to enter the pleural cavity; this, owing to the aspirating influence of inspiration, it would readily do if the smallest hole should remain patent. The young surgeon would do well to practise these operations on the dead body, and familiarise himself with the relations of the various structures they involve.

Some operators of experience completely ignore the absence of peritoneal adhesions, and, even in these circumstances, open the abscess without previous stitching of peritoneal surfaces. The risk and danger of escape of pus into the peritoneal cavity, they hold, is very small if free drainage to the outside is secured. Dr. Neil Macleod considers that, in the circumstances, stitches will not hold in the soft and inflamed liver tissue; he also considers that, in the event of the incision having to be made in the thoracic wall, removal of part of a rib is unnecessary. On account of the liability of the rubber drainage-tube to become nipped when the emptying sac causes a want of correspondence between the wound in the abdominal or thoracic wall and that in the liver, and, also, on account of facility of introduction during the subsequent dressings, this operator uses metal drainage-tubes of suitable lengths—four inches, three and a quarter in., two and a half in., and one and three-

quarter in.—with an oval lumen of four-tenths by three-tenths of an inch. These tubes he introduces by means of a special guide (*Brit. Med. Jour.*, Dec. 26, 1891).

The author's method.—The following easy, rapid, and efficient method of operating on abscess of the liver I have frequently practised, and can recommend. The necessary apparatus (Fig. 43), which can be made by native workmen, consists of a large trocar and cannula (*a*), four to five inches long, by three-eighths of an inch in diameter; a steel stilette (*b*) at least fourteen inches in length; two metal buttons (*c*, *d*) $\frac{1}{4}$ inch at their greatest diameter, with long (half-inch), hollow, roughened necks into which the ends of the stilette fit loosely; six inches of $\frac{1}{2}$ -inch stout drainage tubing (*e*). While the ends of the drainage tubing are held and well stretched by an assistant, they are firmly lashed to the stem of the buttons, over the ends of the shorter (*d*) of which, for additional security, the tubing is also tied (*e*). Two large holes, to provide for free drainage, are then cut close to one end of the drainage-tube. The tube is then mounted on the stilette by inserting one end of the latter through one of the drainage holes and lodging it in the hollow neck of the distal button (*c*), and thereafter so stretching the rubber that the other end of the stilette can be inserted into the neck of the other button (*d*). When thus stretched, the drainage-tube should be capable of passing easily through the cannula. The apparatus being thus prepared and rendered thoroughly aseptic by soaking in carbolic lotion, and the position and depth of the abscess having been carefully ascertained by means of the aspirator and noted, the aspirator is withdrawn and an incision about an inch in length made with a scalpel through the skin at the site of the puncture. The trocar and cannula are then thrust into the abscess and the trocar is withdrawn. After allowing a small quantity of pus to escape, so as to relieve any tension that may be present in the abscess sac, the

stretched drainage-tube, perforated end first, is slipped into the cannula and carried to the back of the abscess. Holding the stretched drainage-tube firmly, and maintaining it carefully in contact with the back of the abscess with one hand, the cannula is withdrawn with the other. Still grasping the

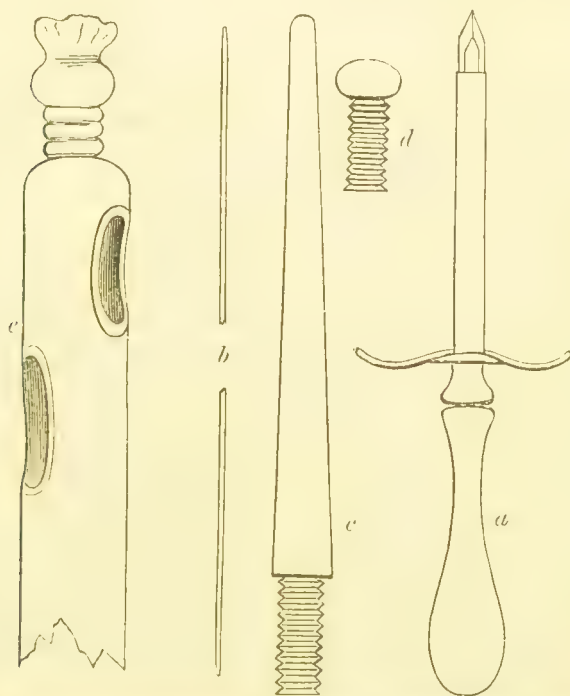


Fig. 43.—Apparatus for operation for Abscess of the Liver. (*a* and *b* reduced.

drainage-tube firmly, the button on the free end of the apparatus is slipped off the stilette, the end of which is made to perforate the drainage-tube close to the button. This it readily does, and the drainage-tube is allowed slowly to resile towards the fixed end, still held in contact with the back of the abscess. When the drainage-tube has completely contracted, the stilette is withdrawn. The drainage-tube is now transfixed with a safety-pin inserted close to the skin, and the superfluous tubing cut off. Pus flows freely

from the tube which now firmly plugs the wound in the abdominal wall and liver, and bridges the peritoneal cavity. When the abscess has nearly emptied itself the usual antiseptic dressing is applied. In operating through the thoracic wall, if deemed desirable, part of a rib may be excised before the trocar is introduced.

I claim for this operation that it is easily done, and that it may be undertaken by the merest tyro in surgery and in the absence of skilled assistance; that there is no risk from bleeding; that, the peritoneum being bridged across by the drainage-tube which is securely grasped by the liver tissue, there is no risk of escape of pus into the peritoneal cavity; that in a very short time lymph is effused around the tube, giving additional security when, after a time, the tube has become loose; that, if deemed necessary, a larger drainage-tube, by stretching it on the stilette in the same way as described, can be substituted for the half-inch tube; that an abscess deep in the liver can be as readily opened, and with as little risk, as one lying near the surface; that the shock is much less than in the cutting and tearing operation; that there is no risk of pneumothorax should the pleural cavity be traversed; and that the drainage obtained is equal to that secured by any other method of operating. Several of my surgical friends have adopted this method of operating, and have expressed great satisfaction with the ease with which it is performed, and with the results.

Other operations. — Some Continental surgeons recommend extensive incision of the liver, using a Paquelin's knife with the view of minimising bleeding. Zancarol, for example, advises that the hepatic and integumental incisions should extend the whole breadth of the abscess cavity, which he mops out and stuffs with iodoform gauze. Certain French surgeons recommend scraping the abscess cavity; most practise irrigation with some antiseptic fluid. These methods do not find favour with English or Indian surgeons.

The method of opening the abscess by caustics, formerly much in vogue, is now abandoned; equally so another method, also formerly employed—namely, that of leaving a rigid trocar in the wound.

Treatment after operation.—For the first few days after a liver abscess has been opened the discharge is considerable, and the dressing may have to be changed frequently. Very soon, however, should the case do well, the discharge rapidly diminishes, and the dressing requires renewal only every other day or every three or four days. During the first week the drainage-tube, provided it be acting efficiently, should not be disturbed, more particularly as it may be difficult to replace. Later, it may be removed and cleaned, and when discharge has practically ceased, cautiously shortened; it is a great mistake, however, to begin shortening the tube before it is being pushed out, or so long as there is any appreciable discharge. If there is the slightest indication, such as rise of temperature, that pus is being retained, the drainage must be rectified and the sinus, if necessary, dilated with forceps and finger, and a full-sized drainage-tube introduced as far as it will go. If this does not suffice, a counter-opening may have to be made. Delay in remedying imperfect drainage is a serious error.

Should an abscess on being opened be found to be septic, or should it become so, it must be flushed out daily, or twice a day, with a weak non-mercurial antiseptic, and a counter-opening made if necessary.

After liver abscess has been opened and is draining well, temperature rapidly falls and, in a few days or almost at once, becomes normal. Should fever persist, it is to be inferred either that the drainage is inefficient, or that there are more abscesses in the liver, or that there is some complication. If it be deemed that there is another abscess, this should be sought for with the aspirator, and, if found, opened and drained. I have seen a patient recover after three abscesses had been so treated.

Treatment of abscess discharging through the lung.—In the case of abscess discharging through the lung, and not progressing favourably, the question of obtaining by surgical means more efficient drainage must be considered. There are two possibilities which render interference desirable. (a) Continued discharge of pus and blood, with or without attendant hectic; a condition which, if it persists, in all probability will in the end kill the patient. (b) Not infrequently prolonged discharge through the lung may induce fibrotic changes in that organ, or may give rise to pneumonia, or to abscess of the lung with all its attendant dangers. In these circumstances it is sometimes difficult to arrive at a decision as to whether an attempt should be made to open and drain the abscess, or to leave it alone. A large proportion of the cases recovers, but at least an equally large number dies. Of the latter, by timely surgical interference, a proportion may certainly be saved.

In all cases of abscess discharging through the lung a careful register should be kept of three things—body temperature, daily amount and character of expectoration, and, once a week, the weight of the patient. If temperature keeps up, if the amount of pus continues the same or increases, or if the patient continues to lose weight, an attempt should be made at all risks to reach and drain the abscess from the outside. If temperature keeps normal, if pus gradually or intermittently decreases, and if the body-weight be maintained or increases, operation is unnecessary, or, at all events, should be deferred.

In exploring the liver in such cases, it must be borne in mind that most likely the abscess cavity is collapsed, and that the sides of the abscess are in contact. Such an abscess is not likely to be discovered unless the needle be thrust in to its full extent, and, whilst a good vacuum is being maintained in the aspirator, slowly withdrawn. If by good fortune the abscess has been traversed, then, when the end

of the needle crosses the cavity, a small amount of pus will be seen to flow. Great care must now be exercised to keep the needle in position so as to serve as a guide in opening the abscess. Recovery has been known to follow the introduction of a drainage-tube in the presumed direction of such an abscess, even although the abscess cavity was not entered, much less drained by the tube.

Treatment of abscess rupturing into a serous cavity.—When there is evidence that an abscess of the liver has ruptured into the peritoneum, into the pleura, or into the pericardium, the particular serous cavity involved must be opened at once and treated on general surgical principles; otherwise, the patient will almost surely die. In the circumstances the surgeon will be justified in assuming great risks.

The *prognosis in early operations* on single abscess of the liver, provided there is no dysentery or other complication, is good. In multiple abscess, or in single abscess if there is active dysentery or other serious complication, prognosis is bad. In multiple abscess, if there are more than two or three abscesses, it is necessarily hopeless.

The *question of return to the tropics* after recovery from liver abscess frequently crops up. If feasible, and if the patient has not to make too great a sacrifice, he ought to remain in a temperate and healthy climate. There are many instances, however, of individuals who have enjoyed permanent good health in the tropics after recovery from liver abscess.

CHAPTER XXVI.

INFANTILE BILIARY CIRRHOSIS.

WITHIN the last fifteen or sixteen years, a peculiar disease of the liver has been noticed in children in Calcutta and, to a less extent, in other large towns of India. It is found to be more prevalent in Hindoo than in Mahomedan children. Thus, in Calcutta, from 1891 to 1893 inclusive, infantile biliary cirrhosis, the name given to this disease, caused 1,748 deaths. Although the Hindoo and Mahomedan populations of that city are about equal, yet as many as 1,616 of the deaths occurred in Hindoos, whilst only eighty occurred among Mahomedans, the balance of the mortality being among the Eurasians and other races. The disease occurs principally in children under one year, rarely attacking those over three years. As a rule, it commences during dentition, or about the seventh or eighth month, running a fatal course in from three to eight months. In rare cases it may commence within a few days of birth. Instead of lasting several months, its progress may be much more rapid, and terminate in death in from two to three weeks.

The cause of infantile biliary cirrhosis is quite unknown. Neither alcohol, syphilis, nor malaria has anything to do with it. The children of the well-to-do are relatively more frequently attacked than those of the poor. It has also been observed that it tends to run in families, child after child of the same parents succumbing within a year or two of birth. In 400 cases (Ghose had only six recoveries: in some of these recoveries the diagnosis was doubtful.

Symptoms.—Commencing insidiously, the characteristic initial enlargement of the liver may have



made considerable progress before the disease is suspected. Nausea, occasional vomiting, sallowness, feverishness, constipation, anorexia, irritability of temper, thirst and languor call attention to the child's condition. On examination, the liver is found to be enormously enlarged, extending perhaps to the umbilicus or even lower. The surface of the organ is smooth; the edge, at first rounded and prominent, as the liver begins to contract becomes sharp and distinct and can be readily grasped between the fingers, the swollen organ feeling hard and resistant. Fever of a low type sets in; the sallowness deepens into profound jaundice; the stools are pale, the urine is dark; and there may be ascites, with puffiness of the feet and hands. Sooner or later, death from cholæmia ensues.

Pathological anatomy and pathology.—

Surgeon-Major Gibbons, who has given an elaborate and most careful account of the pathological anatomy of this disease, concludes that it is a peculiar form of biliary cirrhosis, the consequence of the action on the liver cells of some irritant of gastric origin, which leads to degeneration of the cells in the first instance, with subsequent increase of intercellular connective tissue and, later, of the portal sheaths. The formation of new bile ducts between the hepatic cells, which is a well-marked feature, he regards as evidence of a natural curative effort having for its object a regeneration of the liver cells.

Treatment.—Hitherto, in this disease, treatment has been of little avail. There is some ground, however, for thinking that early removal from the endemic locality, and a complete change of wet-nurse and food, might have a beneficial effect. (*Trans. First Calcutta Med. Cong.*)

CHAPTER XXVII.

PONOS.

UNDER the name "Ponos" Karamitsas (*Gaz. des Hôpitaux*, 1880) and Stephanos (*Gaz. Heb. de Méd.*, 1881) have described a peculiar disease endemic in the islands of Spezzia and Hydra in the Grecian Archipelago. Ponos, in a sense, is analogous to the infantile biliary cirrhosis of Indian cities. Like the latter, it is confined to very young children, it is endemic in particular districts, tends to run in particular families, to pursue a more or less chronic course, and is invariably associated with disease of an abdominal viscus—in this case the spleen. Though not invariably so, it is usually fatal.

Symptoms.—Commencing somewhat suddenly and, commonly, during the first year of life, the earliest symptoms to attract the attention of the parents are languor and a pallor which rapidly acquires a sallow tint. Fever of an irregular character sets in; the spleen enlarges; prostration is very marked; and, it is said, the urine exhales a peculiarly disagreeable odour. Emaciation becomes progressive. Although the digestion is enfeebled and constipation is nearly always present at first, the appetite is fairly preserved, although it may be perverted or excessive. The spleen gradually attains a great size, and is tender—hence the name, "ponos" (pain). The associated fever is of an irregular character, tending to become remittent, the thermometer rising during the exacerbations to 39° or 40° Cent.; towards the end the fever assumes hectic characters. Complications in the form of bronchitis, pneumonia, diarrhoea, dysentery, peritonitis, or meningitis may show themselves. When

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the disease has been established for some time œdema and ascites may occur, and there may be hæmorrhages in or from various organs, especially the gums. In certain cases boils, and even patches of superficial gangrene, are met with. The progress of the disease is very variable ; it may last from one or two months to one or two years.

Pathological anatomy and pathology.—

Of these nothing is known further than that ponos exhibits none of the characteristic lesions of tuberculosis, of leukæmia, or of malaria. Its cause is quite unknown. It has been remarked, however, that it is prone to occur in the children of those who have themselves suffered in infancy in the same way. It is said that in the case of the disease attacking the child of a tuberculous mother, recovery will very promptly set in if the child be supplied with a healthy wet-nurse. It has been suggested that ponos is an expression of malaria, but the *post-mortem* evidences are altogether against this supposition ; moreover, malaria is said to be unknown in the two islands in which this peculiar disease is endemic. The tendency to bleeding from the gums, cutaneous ecchymoses, and liability to hæmorrhages suggest a scorbutic element. Ponos affects the children of rich and poor alike, and those living in good as well as those living in dirty, unhygienic houses.

Treatment.—No specific is known. Quinine, tonics, fruit juices, careful management of the food, and early change of residence would seem to be indicated.

SECTION IV.—INFECTIVE GRANULOMATOUS DISEASES.

CHAPTER XXVIII.

LEPROSY (*Elephantiasis Græcorum*).

Definition.—A chronic infective granulomatous disease produced by a specific bacterium, and characterised by lesions of the skin, nerves, and viscera eventuating in local anæsthesia, ulceration, and a great variety of trophic lesions. After a long course it is almost invariably fatal.

History.—The many allusions in the oldest Chinese, Indian, Syrian, and Egyptian writings to a chronic, disfiguring, and fatal affliction possessing well-marked and characteristic skin lesions, warrant us in concluding that the disease now known as leprosy was as common and familiar in the East in times of remotest antiquity as it is at the present day.

There is some evidence—necessarily of a negative character—that leprosy is of comparatively recent introduction into Europe. The earlier Greek and Latin writers do not mention the disease. Hippocrates, who, had he been practically acquainted with leprosy, would undoubtedly have described it accurately and fully, makes but brief allusion to the subject. Aristotle is the first of the Greek writers to give an unequivocal description. We may infer, therefore, that the introduction of leprosy into Greece probably took place between the time of Hippocrates and that of Aristotle—that is to say, between 400 B.C. and 345 B.C. Most likely it came from Egypt. In the time of Celsus—53 B.C. to A.D. 7—it was still a rare disease in Italy ; but, during the earlier centuries





of our era it increased there, and, probably following in the wake of the Roman conquests, it appears to have spread thence over the greater part, if not over the whole, of Europe. By the end of the seventh century it was well known in Spain, France, and Lombardy. There is a notice of its occurrence in Ireland in 432. As regards England, the first allusion to leprosy refers to about the year 950. The popular idea that it was brought to this country from the East by the returning Crusaders (*circa* 1098) is therefore incorrect; though, doubtless, the Crusaders, and the multiplicity of pilgrimages so much the fashion in the Middle Ages, and the destitution arising from the many wars of the period, had something to do with its rapid diffusion and great increase about this time.

So common was leprosy during the Middle Ages that the rulers and clergy of nearly all European States, becoming alarmed at its rapid extension and terrible ravages, took measures, by instituting leper asylums and enacting stringent laws for the segregation and isolation of lepers, to restrict the spread of what was speedily becoming almost a general calamity. These measures, based on what we now know to be a correct appreciation of the infectious nature of the disease, were ultimately crowned, in the case of most European countries, by almost complete success. Reaching its acme during the fourteenth century, leprosy then began gradually to decline, although as regards Great Britain it did not finally disappear as an indigenous disease until the end of last century. It died out first in England, later in Scotland—the last British leper dying in Shetland in 1798. In Italy, France, Spain, Germany, and Russia the repressive measures were almost equally successful, although in these countries, in Greece, and in the Greek islands leprosy of indigenous origin is still occasionally to be seen. The only country in Northern Europe in which at the present day it may be said to linger to any extent is Norway, where, in places, it is still by no

means uncommon (in 1890 there were 1,100 known lepers); but even here, under a system of segregation and comparative isolation—more humane perhaps in its application than that practised by our ancestors, although identical with it in principle—the disease is rapidly dying out.

Apart from Egypt, we know nothing of the early history of leprosy in Africa. We are equally ignorant as regards aboriginal America. The historians of the Spanish conquest do not allude to it as a native disease; we appear, therefore, to be justified from this circumstance in concluding that the introduction of leprosy into the New World was probably effected through the negroes in the days of the slave trade.

Rise of modern knowledge of leprosy.—The more important landmarks in our modern knowledge of leprosy are, first, the publication in 1848 of Danielssen and Boeck's *Traité de la Spedalskhed*, in which, for the first time, the clinical features of the disease were carefully and critically described; second, the descriptions of the macroscopic and microscopic lesions—the leproma, the nerve lesions, and the lepra cell—by Virchow, Vandyke Carter, and many others; and last, the discovery in 1874 by Armauer Hansen of the specific cause of leprosy—the *Bacillus lepræ*—a discovery which brings this disease into line with tubercle, and which has given a much-needed precision to our ideas on the important subjects of heredity and contagion, and on other practical points bearing on the question of the leper as a source of public danger and on his treatment and management.

Geographical distribution.—Whatever may have been the case formerly, at the present day, with unimportant exceptions, leprosy is a disease more particularly of tropical and sub-tropical countries. So generally is it diffused in these that it would be more easy to specify the tropical countries in which leprosy has not, than to enumerate those in which it has, been ascertained to exist. Moreover, it is probable that in many of the countries not yet





positively known to harbour the disease it does really exist; for experience shows that the endemic area of leprosy enlarges as our knowledge of the natives of the uncivilised regions of the earth becomes more intimate. It may be safely concluded, therefore, that with the exception of a few insignificant islands, leprosy is an element, and often an important element, in the pathology of nearly all warm countries.

The only tropical country of any magnitude about which we have anything like accurate leprosy statistics is India; and even in this instance the figures, for many reasons—principally errors in diagnosis and concealment—are untrustworthy. According to the census of 1891, after making allowance for error, it is estimated that in British India there were 105,000 lepers in a population of 210,000,000—a ratio of about 5 in 10,000. Respecting China, of all countries probably the one in which there is the largest number of lepers, we have no figures to go by; but, judging from what is seen in the coast towns and treaty ports, the number of lepers there is even greater than in India. In Japan, in the Philippines, in Cochin China, in the Malay peninsula, in the islands of the Eastern Archipelago, and in several of the South Pacific islands, in Persia, Arabia, and Africa, the disease is common enough. The same may be said of the West Indies and of the tropical regions of America.

As regards more temperate countries, we know that there is a considerable number of lepers at the Cape, a few in Australia (principally Chinese but also a few Europeans), a few in San Francisco (Chinese). In Canada and in the United States there are also a few lepers of European blood, but their number is quite insignificant. In New Zealand, where leprosy was at one time common among the Maoris, it has died out. There are a good many lepers in Iceland. It is also reported as existing among the aborigines of the Aleutian peninsula and

Kamschkatka. In Great Britain and other European countries, particularly in the capital cities, lepers are not infrequently exhibited at medical societies; but, with rare exceptions, these cases are not of indigenous origin, most of them having contracted the disease abroad.

Though thus extensively diffused, leprosy is by no means equally prevalent throughout the wide area indicated. Thus in China it is comparatively rare in the northern provinces, excessively common in the southern. In India a similar caprice of distribution is noted; in Burdwan, for example, the proportion of lepers in 10,000 of the population is as high as 19·5, whereas in several other districts it is as low as 1·5, or even lower. This caprice of distribution does not seem to depend on climate, geological formation, or suchlike physical conditions; leprosy is found in mountainous districts, on the plains, on the coast, in the interior, in all varieties of climate, and on all kinds of geological strata. Social conditions, it would seem, have most to do in determining distribution; its endemic prevalence appearing to be bound up in some way with uncleanly habits, squalor, dirt, and poverty—not, be it noted, directly caused by these things, but associated with them.

Recent introduction.—An interesting and, from the ætiological standpoint, an important circumstance about the geographical distribution of leprosy is its appearance and rapid spread in recent times in certain islands whose inhabitants, there is good reason to believe, had previously been exempt. This modern introduction of leprosy into virgin soil, so to speak, has taken place in two islands at least—the Sandwich Islands and New Caledonia.

Sandwich Islands.—In the case of the Sandwich Islands leprosy was noted among the aborigines for the first time in 1859. After the most painstaking investigation Dr. Hildebrand failed to trace it farther back than 1848. Soon after its presence was recognised the disease spread so rapidly that by the year



1865 there were 230 known lepers in a population of 67,000. By 1891 the native population, from various causes, had diminished to 44,232; of these 1,500 were lepers—about 1 in 30.

New Caledonia.—In New Caledonia (Grall, *Arch. de Méd. Navale*, Oct., Nov., Dec., 1894) leprosy was unknown until 1865. It is supposed to have been introduced about that time by a Chinese; the man was well known. Its rapid diffusion throughout the island can be, and has been, traced step by step. In 1888 the lepers numbered 4,000.

Isle of Pines.—Similarly, in the Isle of Pines it was introduced in 1878, and has since spread. In the Loyalty Islands the first case was seen in 1882; in the island of Mare alone in 1888 there were 70 lepers.

Symptoms.—Although, as will afterwards be explained, the *Bacillus lepræ* is the cause of all leprosy, the clinical manifestations of its presence are far from being identical in every case; indeed, they are almost as varied as are those of syphilis or of tubercle. Our early conceptions of the disease, derived for the most part from the Bible or poetical literature, in which the leper is symbolical of all that is loathsome and hopeless, are apt to mislead. As a matter of fact, in its earlier stages leprosy is far from being always, or even generally, a striking disease. Often for years the only visible evidence of its existence may be two or three small blotches, or perhaps one or two patches of pale or pigmented skin on trunk or limbs—very likely concealed by the clothes and perhaps disregarded by the patient himself—the true significance and nature of which can be appreciated only by the expert. It is generally not until the later stages that we see the disfiguring and extensive lesions on which the popular conception is founded. As a rule, leprosy is a disease of very slow development. Sometimes, it is true, it is suddenly and frankly declared from the outset, and progresses rapidly; but, in the vast majority of cases, the early

lesions are trifling and are apt to be misinterpreted and overlooked, and years elapse before serious mutilation or deformity is produced. The student must bear this important practical fact in mind in the study and diagnosis of all equivocal skin lesions in persons residing in, or coming from, the endemic haunts of leprosy.

To facilitate description, it seems advisable to divide the evolution of leprosy into stages, premising, however, that the division proposed is in great measure an artificial one. What are here designated stages are not, all of them, in nature and in every instance, abruptly separated or even present; for the most part they merge imperceptibly into each other and, in not a few instances, some of them cannot be recognised.

1. The primary infection.
2. The period of incubation.
3. The prodromata.
4. The primary exanthem or macular stage.
5. The period of specific deposit.
6. The sequelæ — ulceration, paresis, trophic lesions.
7. Terminations.

1. *The primary infection.*—Seeing that leprosy is caused by a specific germ, there must have been a time in the history of every leper when the infecting germ entered the body. In the case of many specific diseases, such as syphilis, the site of the primary infection is indicated by a well-marked local lesion and the time of infection can usually be ascertained. So far as present knowledge goes, this much cannot be affirmed of leprosy; in this disease we know of nothing that indicates precisely either the seat or, with rare exceptions, the time of primary infection. In this respect leprosy resembles tuberculosis. We are equally ignorant as to the condition of the infecting germ, whether it enters the organ or organs through which it gains access as spore or as bacillus; and, also, as to the medium in or by which it is conveyed.



We cannot say whether it enters in food, in water, in air; whether it passes in through the unbroken epithelium, or whether it is inoculated on some accidental breach of surface, or, perhaps, introduced by some insect bite.

Recently Sticker found *Bacillus lepræ* in the nasal mucus in 128 out of 153 lepers examined. He considers that the initial lesion of the disease is a specific ulceration of the cartilaginous septum of the nose; this lesion persists and is an active source of infection during life. Of this condition epistaxis, he maintains, is often an early symptom.

Although we are in absolute ignorance as to the process of infection, we may be quite sure that in leprosy there is an act of infection, and that the infective material comes from another leper. Leprosy has never been shown to arise in a country *de novo*. There are many facts and arguments to support this statement; their discussion is deferred until the important practical subjects of heredity, contagion, and the hygienic questions connected therewith, come to be considered.

2. *The period of incubation*.—This is generally, possibly always, long, and has to be reckoned usually in years—two or three at least. There are cases on record in which the period of incubation must have been longer even than this. Danielssen mentions one in which the period was ten years. Leloir describes another in which fourteen years or more, and Höegh one in which twenty-seven years, elapsed between the time at which infection was presumed to have occurred and the first manifestations of the disease. On the other hand, cases are on record in which the incubation period was set down at three months or even at a few weeks.

3. *Prodromata*.—Fever of greater or less intensity, and occurring more or less frequently is, almost invariably, a feature of the prodromal stage of leprosy. Febrile attacks may recur off and on during one or two years. It is well to bear in mind that in tropical

countries such attacks are apt to be looked on as malarial. Another very common prodromal feature is an unaccountable feeling of weakness, accompanied usually by a sensation of heaviness and a tendency, it may be irresistible, to fall asleep at unusual times. Dyspeptic troubles, associated with diarrhœa in some cases, with constipation in others, and usually attributed to "liver," are also common. Epistaxis and dryness of the nostrils, corresponding to the epistaxis in the prodromal stage of typhoid, tuberculosis, and such as is sometimes met with in early syphilis, are noted by Leloir. Headache; vertigo; perversions of sensation—such as localised pruritus, hyperæsthesia, "pins and needles," neuralgic pains—intermittent for the most part, and perhaps very severe and especially common in the limbs and face; general aching, rheumatic-like pains in loins, back, and elsewhere; all or any of these for months may herald the explosion of leprosy. They point to a direct and early implication of the nervous system by the *lepra bacillus*, or to toxin poisoning by its products.

Another curious feature in early leprosy, also noted by Leloir, is the liability in many instances to excessive sweating, which comes on without apparent, or on very slight, provocation. I had once under observation an Englishman who subsequently developed leprosy, in whom this prodromal symptom was particularly pronounced, so pronounced that he had remarked it himself. This gentleman for many years kept a diary in which, among other things, he recorded very carefully matters relating to his health; so that it was easy to trace from this diary the gradual evolution of his leprosy. The first unequivocal manifestation of the disease, an extensive erythematous patch on the ulnar side of the left arm and hand, which afterwards became anæsthetic, and two or three pigmented spots on the cheek, back, and leg, was noted on March 3rd, 1894. Five years before this there is an entry in his diary, under date June 4th,





1889, of the first of a long series of severe headaches, transient febrile attacks, and progressive deterioration of health and vigour. Under date of June 9th, 1892, is the first mention of the profuse perspiration which, occurring without obvious cause, continued to be a prominent feature until several months after the appearance of the skin lesions referred to. In the diary such entries as the following frequently occur: "Nov. 20th, 1892.—Feel poorly and sweating inordinately; wondering what is coming over me. I am very ill." "Dec. 5th to 19th, 1892.—Ill in bed. I had furious sweats during this time, and had dreadful pains in the back, lower part." And again: "Dec. 21st, 22nd.—Feeling very ill; awful sweats; weak; done up." As pointed out by Leloir, this hyperidrosis may be general or it may be confined to particular parts, generally the trunk, the limbs being unaffected or even being the subject of anidrosis. A still more limited anidrosis is sometimes noted; it usually happens that these non-sweating spots become anæsthetic at a later period of the disease.

Just as in syphilis, in a very small proportion of cases of leprosy there is a complete absence of constitutional symptoms prior to the appearance of the specific skin eruption.

4. *The primary exanthem.*—After a longer or shorter period of indifferent health, and being sometimes preluded by an outburst more severe than usual of fever and other prodromic phenomena, an eruption appears on the skin. The occurrence of this generally coincides with, or is soon followed by, an improvement in the general health.

Although strictly macular, this eruption—the primary exanthem of leprosy—varies in different cases both as to the size of the spots, their number, duration, and other characteristics. They may be no larger than a millet seed, or they may occupy surfaces many inches in diameter; they may be numerous, or there may be only two or three. The earlier spots

are usually purely erythematous, disappearing on pressure, and being darkest in the centre and shading off towards the periphery. But in some cases they may be pigmented from the outset ; or they may be mere vitiliginous patches ; or all three forms of macula may concur in the same individual—erythematous, pigmented, and vitiliginous. In not a few lepers what in the first instance was an erythematous patch may in time become pigmented, or it may become pale ; in the latter case the loss of pigment is usually associated with a certain degree of atrophy of the cutis. Or it may be that the centre of an erythematous patch clears up, the periphery of the patch remaining red and perhaps becoming pigmented ; so that the affected spot comes to present the appearance of a red or dark ring, or portion of a ring, enclosing a patch of pale, usually anæsthetic skin. In certain instances the eruption of the various forms of maculæ may be preceded by local paræsthesiæ, such as a sense of burning, tingling, pricking, and so forth.

At first the maculæ may be evanescent and may fade wholly or in part in the course of a few days, weeks, or months ; but as the disease progresses, and fresh spots appear, they tend to greater permanency, to be more liable to pigmentation, and are partially or wholly anæsthetic from the outset, or subsequently become so.

A striking feature in this and in all leprous eruptions is the loss of the hair in the affected areas. Another striking circumstance in this connection is the fact that the most hairy part of the body, the scalp, is never affected either with leprous eruptions or with what could be considered leprous alopecia. As the face, particularly the superciliary region, is prone to all forms of leprous eruption, falling of the eyebrows is a very usual, very early, and very characteristic phenomenon. The beard, too, is apt to be patchy, particularly in nodular leprosy. In many instances, before they drop out, the individual hairs become white, or downy, or splintered, or monillated.



The most frequent seats of the primary macular eruption are the face, especially the superciliary region, the nose, cheeks, and ears; the extensor surfaces of the limbs; the backs of the hands; the back, buttocks, abdomen, and chest. The palms of the hands and the soles of the feet are rarely if ever attacked; the scalp is never attacked. At this stage of the disease the mucous membranes are very rarely affected.

In the distribution of the maculæ a rough symmetry may or may not be discernible.

5. *The period of specific deposit.* — During the stage just described, if there be any thickening of the skin or other evidence of new growth, it is barely perceptible to sight and but slightly to touch. Sooner or later, however, another stage is entered on, a stage characterised by the deposit or, rather, growth of a tissue possessing well-marked specific characters. This deposit occurs either in the skin, or in the continuity of the peripheral nerve trunks, or in both. If in the first situation, nodular or, as it is sometimes called, tuberculous leprosy is the result; if in the second, we have nerve or anæsthetic leprosy; if in both of these situations, then what is known as “mixed leprosy” is produced. These three forms of the completely developed disease, though having much in common, are, as a rule, clinically fairly distinguishable. It is customary, therefore, to describe them separately.

NODULAR LEPROSY.

This form of leprosy often appears without a well-marked preliminary macular stage, being ushered in, after a longer or shorter prodromic stage, by a smart attack of fever and the rapid development on the face or elsewhere of the specific lesion. In other instances a well-defined but, in comparison with nerve leprosy, short macular stage precedes the appearance of the characteristic lepromata (Fig. 44).

The essential element in nodular leprosy is the

leproma. The dimensions, the combinations, the situations, the growth, and the decay of this give rise to the more manifest symptoms of the earlier stages, at all events, of the disease. The leproma, which will be more fully described in the section on the pathological anatomy of leprosy, is formed by infiltration of the deeper layers of the derma with what at first is a small-celled, somewhat dense neoplasm. As this slowly or more rapidly increases it forms a prominent, rounded boss or protuberance covered with unbroken epidermis. In size it ranges from the dimensions of a split pea, or of a bean, to a great plaque many inches across. In colour it is different according to its age and condition, and according to the natural hue of the skin of the leper; it varies from red to dirty pink in the earlier and congestive active stage, to dark brown or dirty yellow in the later stages. It is generally, though not always, especially at first, anæsthetic to some degree, if not absolutely so; it is devoid of hair, usually somewhat greasy-looking and, perhaps, stippled with gaping follicles. Though not so hard as keloid growth, it is fairly firm to the touch, and, unless very extensive, can be readily raised up and freely moved over subjacent structures. Isolated lepromata are usually round or oval; when contiguous they may coalesce, forming patches of irregular outline.

When many lepromata run together, or are closely set, the growth causes the natural folds of the skin to be exaggerated; and thus great disfigurement, especially of the face, may ensue. Thus the skin of the forehead and eyebrows—an early and favourite site of leprosy infiltration—is thrown into massive folds and overhangs the eyes; the fleshy parts of the nose broaden out; the cheeks become massive; the lips are thickened and protrude; the chin is swollen and heavy; the external ears are thick and pendulous; and the bloated, dusky, wrinkled, greasy, passive countenance acquires the repulsive appearance very appropriately designated “leontiasis.”





Fig. 44.—NODULAR LEPROSY (*Jeloir*).

Nodules may appear in greater or less profusion on the limbs and body; favourite sites being the backs of the hands, the external surfaces of the arms, wrists, thighs, and the groins. On the trunk they may occupy very large areas, forming extensive plaques. As a rule, in the latter situation they are not so prominent as on the face and arms. The same remark applies to the legs, where the infiltration is usually dusky, diffuse, ill-defined, and prone to ulcerate.

From time to time, and at longer or shorter intervals, fresh tubercles appear, their formation generally concurring with an outburst of leprous fever. Occasionally, and this is very often observed during an intercurrent attack of some acute disease—such as an exanthematous fever, or erysipelas, or even of some exhausting disease like phthisis—all or a proportion of the nodules are temporarily absorbed, leaving only slight traces behind. But the normal and usual fate of the nodule is either first to soften in the centre and then to be absorbed, leaving a smooth circular patch of scar tissue; or, after softening, to ulcerate and discharge a sticky, yellowish pus. This discharge tends to dry up into crusts, ulceration proceeding underneath. Finally the ulcer may heal, leaving an irregular, depressed scar.

When the septum of the nose is affected, the cartilage breaks down, the tip of the organ becomes depressed, and a stinking discharge escapes from the nostrils. In such circumstances breathing is very much interfered with, more especially if, at the same time, leprous deposit occurs or ulcerates in or about the glottis, the epiglottis, pharynx, tongue, or mouth generally. The senses of smell and taste are then lost for ever.

The eyes, also, are sooner or later attacked, lepromatous growth spreading from the conjunctiva on to the cornea or into the anterior chamber, or originating in the iris or ciliary body. Ultimately this organ also is destroyed, and with it the sense of sight.

Thus, in time, with the exception of that of hearing, one sense after another disappears. Ulcers form





everywhere from the breaking-down of the nodules or from injuries to the insensitive skin. The cervical and inguinal glands, owing to leprous infiltration, swell and perhaps suppurate and become fistulous; the abdomen enlarges from leprous, perhaps combined with amyloid, infiltration of the liver, and there may be diarrhœa from amyloid disease of the intestine. In addition to these troubles, if the patient live, the nerve trunks are attacked, and then the neuralgic, parietic, and trophic lesions of nerve leprosy are superadded. The fingers and toes ulcerate and drop off, or they become distorted and atrophied; or the phalanges are absorbed, the hands and feet becoming reduced to useless stumps. A peculiar goat-like smell is emitted by the ulcerating, decaying body. Altogether, the blind, lame, unhappy wretch—still retaining his intellect but devoid of every sense except that of hearing, breathing with difficulty through a stenosed larynx, and racked by neuralgic pains and irregular outbursts of fever—comes to present, before the inevitable death from exhaustion occurs, a sadder, more loathsome, and more repulsive picture than imagination could conceive. Fortunately, in a large proportion of cases, the leper is mercifully carried off by phthisis, pneumonia, or some intercurrent affection at an earlier period, and before his disease could be said to have run its full course.

NERVE LEPROSY.

Just as in nodular leprosy, in nerve leprosy the prodromic and macular stages may be severe, or slight, or altogether absent. Usually, however, in nerve leprosy, much more frequently than in nodular leprosy, the ulterior and more distinctive lesions are preluded by a long and well-marked macular stage, during which large areas of skin are occupied by erythematous (Fig. 45), by pigmented, or by vitiliginous patches. The ringed form of eruption is a very usual one; a red, congested, slightly elevated

and, perhaps, hyperæsthetic border enclosing a larger or smaller area of pale, anæsthetic, non-sweating integument—the whole resembling somewhat one of those extensive body-ringworms so common in natives of hot, damp climates, for which these rings are sometimes mistaken. Such eruptions may come and go, or they may be permanent, or they may spread and multiply during many years before the more distinctive and graver signs of nerve leprosy are evolved.

A frequent and very distinctive symptom of this type of the disease, occurring often about this time, is the sudden appearance of bullæ (*pemphigus leprosus*)—one or more or a series of them—on the hands, feet, knees, backs of thighs, or elsewhere. These bullæ vary in size from a pea to an egg. After a few days they rupture, exposing a reddish surface which presently crusts over, exfoliates, and finally turns into a pale, perhaps anæsthetic, spot with a sharply defined, pigmented border. More rarely the site of the bulla ulcerates. Should similar bullæ be formed in the neighbourhood of the first, the resulting ulcerations may unite into an extensive, probably superficial, serpiginous-looking sore.

A time comes when evidence of profound implication of the nervous system, in the shape of severe neuralgic pains, formication, hyperæsthesia, or anæsthesia, becomes more accentuated. The lymphatic glands enlarge, and there is often considerable fever and general distress. Hitherto, the most prominent symptoms have been the skin lesions. These may remain or even increase; on the other hand, they may in part or entirely disappear. But whether the skin lesions increase or retrograde, evidences of profound implication of the peripheral nervous system now distinctly show themselves; the neuralgic pains still further increase, and hyperæsthesia, anæsthesia, and various paræsthesiæ, along with trophic changes in skin, muscle, and bone, the results of nerve destruction, become the dominating elements in the case.







Fig. 45.—NERVE LEPROSY (*Leloir*).

If at this stage the ulnar nerve where it passes round the internal condyle of the humerus be examined, generally it will be found to be the seat of a fusiform swelling perhaps as thick as the little finger. Other nerves, such as the anterior tibial, the peroneal, more rarely the median, radial, brachial, and cervical nerves, especially where they pass over a bone and lie close under the skin, can be felt to be similarly swollen. Occasionally even the smaller nerves, where superficial, can also be detected hard and cord-like. At first these thickened nerves are tender on pressure, and the parts they supply may be the seats of hyperæsthesia and acute neuralgia. By degrees the great thickening of the nerve trunks decreases somewhat, the hyperæsthesia and neuralgia subside, and anæsthesia, paresis, muscular atrophy, and other trophic changes take their place. For a time the condition may fluctuate; the neuritis apparently may come and go with corresponding changes in the condition of the area subserved by the affected nerves. Sooner or later, however, fibrotic changes ensue in the neural leprous deposits, and the nerve tubules ultimately atrophy and disappear. The nerve tissue is now irreparably damaged, and trophic changes steadily advance. In other instances anæsthesia comes on without neuralgic pains, without hyperæsthesia, without constitutional symptoms, without discoloration of the skin, the patient discovering its existence by accident.

In nerve leprosy the anæsthesia begins most commonly in the feet, the thighs, hands, arms, forearms, and face. Later, and more rarely, it affects the trunk. The anæsthesia, though associated with well-marked lesions of the larger nerves, does not always, or even as a rule, coincide accurately with the anatomical distribution of their terminals; a circumstance which tends to show that the anæsthesia is not always and simply the result of lesion of nerve trunks, but that it may be the effect of the destruction by the bacillus of the nerve terminals,





themselves; a suggestion which is strengthened by Gerlach's discovery that in anæsthetic leprosy the bacilli appear first in the skin around the nerve terminals, and only subsequently extend upwards to the nerve trunks. Another, and sometimes a very striking, fact in nerve leprosy is the symmetry observed in the distribution of some of the anæsthetic areas. This symmetry is by no means invariable; in not a few cases, however, it is very perfect and remarkable.

At the outset the anæsthesia in the affected patches may not be absolute; it may also come and go; and it may be very superficial, deep pressure being for a long time appreciable. But when the anæsthesia becomes, as it were, settled in a part, it seems gradually to extend deeper into the tissues; so that after a time it is absolute, and the parts may be pinched, incised, and even seared with fire, and the leper be absolutely unconscious of pain or even of being touched.

Step by step with the progress of the anæsthesia, atrophy of the subjacent muscles supplied by the thickened nerves proceeds. Along with the atrophy there is a corresponding distortion and a corresponding loss of power. There is no ataxia or inco-ordination of movement—simply feebleness. Thus the forearm wastes, the grasp is weakened, the thenar and hypothenar eminences, and the interossei melt away, and the *main-en-griffe* or some such deformity is gradually produced (Fig. 46). Similar changes occur in the legs and feet, so that the power of walking is much impaired. The muscles of the thighs and upper arms, the pectorals, and the muscles of the face follow suit very much as in progressive muscular atrophy, only in the latter disease there is no superjacent anæsthesia.

In the affected nerve areas all the muscles are not simultaneously or equally attacked, so that, especially in the face, curious distortions may ensue. These facial atrophies, whether symmetrical or one-sided,

in time produce a facies as characteristic of nerve leprosy as leontiasis is of nodular leprosy. Owing to muscular atrophy the eyes after a time cannot be closed; the upper lid droops, the lower lid becomes everted, and the eye itself may become fixed. At first, owing to exposure of the organ, there is lachrymation; but by-and-by the secretion of tears dries up, the congested conjunctiva becomes cornified, the cornea ulcerates or turns leucomatous, and in the end sight is entirely lost. Ulceration often occurs in the mucous membrane of the nose, the septum being destroyed as in the nodular form; the tip of the nose may then fall down or be entirely lost. The lips, too, may become paralysed, thereby interfering with articulation and permitting the saliva to dribble from the mouth in a constant stream. Changes occur, too, in the mucous membrane of the mouth; the gums may retract, exposing the maxillary bone, the teeth ultimately dropping out. Anæsthesia of the tongue and buccal mucous membrane, and implication of the muscles of mastication may render eating and articulation very difficult.

In time the skin of anæsthetic patches on the limbs tends to atrophy; it loses its glands and hairs, and, in the end, may become so thinned and tense that it may actually burst into long cracks. The nails are not generally shed, but they become rough, or thinned, or atrophied into minute, hook-like appendages.

Ulcers form over exposed parts of the hands and feet. They may penetrate and disorganise the joints and thus often cause fingers and toes to drop off, one after another. Or, perhaps, an abscess forms around a phalanx, destroys the periosteum, and ultimately leads to loss of the bone. Or a sort of dry gangrene may amputate finger or toe. Or there may be a curious interstitial absorption of one or more phalanges, the shaft of the phalanx wasting more rapidly than the articulating surfaces. In any of these ways the fingers and toes are distorted or







Fig. 46.—NERVE LEPROSY: MAIN-EN-GRIFFE (Leloir).

destroyed. It is no unusual thing to see on a leper's hand a finger in which one or more of the phalanges have been thus got rid of without destruction of the fleshy part, or with only a general shrinking of this. Thus it comes about that a distorted, talon-like nail may crown a finger which is a mere stump; or, the finger having been entirely absorbed, the nail springs, as it were, directly from the knuckle.

Perforating ulcer of the sole of the foot, usually under the ball of the great toe or the heel, is a very common lesion in nerve leprosy.

On the whole, the advance of this form of leprosy is much slower than that of the nodular variety. The average duration of the latter is from eight to nine years, of nerve leprosy about eighteen years. Often such lepers live much longer—twenty, thirty, or even forty years. As can readily be understood, the end of these cases is quite as sad and repulsive as that of nodular leprosy. Death seldom results directly from the disease itself; diarrhœa, chronic nephritis, phthisis, pneumonia, or bronchitis being, one or other of them, usually the immediate cause of death.

MIXED LEPROSY.

As already explained, in most cases of nodular leprosy trophic changes from implication of nerve trunks ultimately supervene. Similarly, though not so frequently, nodular infiltration of the skin may appear in the course of what originally seemed to be a case of pure nerve leprosy. In yet other cases nodular and nerve lesions concur from the outset. In one or other of these ways what is known as mixed leprosy is produced. The lesions are in no way different from those already mentioned, and therefore this form of the disease does not call for more detailed description.

Pathological anatomy. — *Bacillus lepræ.* — The lesions of leprosy are the result, direct or in-





direct, of the proliferation of the *Bacillus lepræ* in the tissues. This parasite (Fig. 47) in size, shape, and staining reactions closely resembles the bacillus of tubercle. In length it is from half to two-thirds, and in breadth about one-sixteenth the diameter of a blood corpuscle. The ends of the rod—which is always straight—are in many specimens somewhat



Fig. 47.—*Bacillus lepræ*. $\times 1000$ (Muir and Ritchie).

attenuated; and in many instances—presumably in old bacilli—a moniliform arrangement of the protoplasm, as if from spore formation or, according to Hansen, from disintegration, can be detected. By some authorities it is said to possess a gelatinous capsule. In common with *Bacillus tuberculosis* and *Bacillus smegma* it retains carbol-fuchsin stains after being treated with mineral acids. It may be distinguished from *Bacillus tuberculosis* by its staining more readily with cold weak solution of carbol fuchsin, and by being decolourised more easily with dilute acids; by the impossibility hitherto experienced of growing

it on the usual or on any culture media, and of inoculating it in man and the lower animals; by its tendency to occur in dense clusters and in greater numbers; and by its very generally being found inside cells or, according to Unna, in zoogloea masses in the lymphatic spaces.

Specimens of the bacillus can be procured readily by excising a portion of a leproma—a proceeding, in consequence of the absence of sensation in most tubercles, not usually much objected to by lepers; or they may be obtained by including a succulent leproma in a pile clamp, slowly screwing up the jaws of the instrument so as to drive out the blood, pricking the now pallid leproma, and then collecting on a cover-glass the droplet of “leper juice” which exudes from the puncture. The juice may be spread out on the cover-glass, fixed, stained, and decolourised as for the demonstration of tubercle bacilli; or it may be examined fresh. Better preparations are made by making with a small scalpel a minute incision into the compressed leproma scraping some of the tissues from the under surface of the skin, and smearing this with the juice on to the cover-glass. One must be careful to exclude the possibility of contamination with *Bacillus tuberculosis*, with which lepers are often infected.

If examined fresh, or if a morsel of leproma be teased up in water, the bacilli may be seen both inside and outside the cells and in active motion. Whether this motion is simply molecular, or whether it is vital, it is hard to say; probably the former, for, whilst osmic acid does not stop it, it is immediately arrested by the addition of viscid fluids, such as glycerine or albumin water.

The bacillus is found in all primary leprous deposits; in the skin leproma—where it occurs in prodigious numbers; in the meagre infiltration of the macular eruptions—where it is much more sparsely distributed; in the early stage of leprous neuritis—where, also, it is present only in small numbers; in





the specific lesions of the liver, spleen, testes, and lymphatic glands. In the blood-vessels it has been found in the endothelium and, occasionally, free in the blood or enclosed in leucocytes. It is abundant in the purulent discharges from ulcerating lepromata, or other forms of primary leprous infiltration. It has very rarely been found in the spinal cord or in the lungs. It is doubtful if it occurs in the brain, the intestinal tract, or in the kidneys, although the latter are prone to inflammation in leprosy. It is not found in muscle, in bone, or in cartilage; and it is not necessarily present in the secondary trophic lesions of nerve leprosy, or in secondary inflammatory effusions.

The leproma.—The young leproma presents a smooth, white, glistening section. When the leproma is older the cut surface has a brown tint, and the morbid tissue may become, from fibrotic changes, harder, or, from degeneration, softer. The specific lesion of leprosy differs from that of tubercle, inasmuch as it is well supplied with blood-vessels, contains no giant cells (Hansen), and never undergoes caseation. If hardened, cut, stained, decolourised, and examined under the microscope, the leproma is found to consist principally of small round cells about the size of a leucocyte, epithelioid cells, and fusiform cells—the two latter in increasing numbers with the age of the leproma. It can be seen that these cells have infiltrated and partially dissociated all but the most superficial layer of the derma. It may be further observed that the cells are arranged for the most part in groups, generally around and near blood-vessels; and that a very large proportion of them contain bacilli, some cells having only a few, whilst others are literally crammed with the organisms. Isolated bacilli are also found scattered through the preparation, apparently free in the lymph spaces. The bacilli are never seen inside the nuclei of the implicated cells.

In addition to the bacilli-bearing cells, and increasing in number with the age of the lesion, a number of brown granular bodies, larger and smaller,

which have been named "globi," are to be found. These Hansen holds to be cells in which the bacilli have perished and become granular. It is to them that the brown colour of old lepromata is due.

There has been considerable discussion as to the exact position of the bacilli as regards the lepra cells—whether they lie inside the cells or whether they are free. On the one hand, Unna holds that they lie free in the lymph spaces, and that they are never in the cells, the appearance of cell inclusion being produced by the zooglœa arrangement so common with bacteria. On the other hand, Leloir maintains that some of the bacilli are free, whilst others are inside the cells. A third set of observers, following Hansen, holds that the bacilli are almost invariably included within cells, the nuclei of which can readily be demonstrated surrounded by the parasites.

Other lesions.—The histology of the infiltrated macula is practically the same as that of the leproma, the number of bacilli, lepra cells, and globi being proportionately fewer. In old maculæ, as in very old lepromata, the bacilli may be hard to find or entirely absent. In the anæsthetic maculæ the terminal nerve fibres are degenerated.

As the fusiform thickening of the larger nerve trunks in nerve leprosy is a secondary inflammation, bacilli may not always be found in it, although at the very commencement of the nerve disease bacilli, both in cells and, according to Leloir, free between the nerve tubules, are present and may even lie in the nerve tubules themselves. In time the affected nerves become mere fibrous cords destitute of nerve tubules.

The anatomy and histology of the various trophic lesions are such as are found in other examples of destructive neuritis, and are in no way peculiar to leprosy; they do not, therefore, call for description here.

In nodular leprosy the liver and spleen are the subjects, in many instances, of a peculiar infiltration which, in well-marked examples, may be visible to the





naked eye. Fine yellowish-white dots and streaks are seen to occur in the acini of the former. These dots and streaks consist of new growth in which bacilli abound ; according to Leloir, the parasites are never found in the hepatic cells themselves.

In all cases of nodular leprosy the testes atrophy and undergo fibrotic changes, bacilli and globi being found both in and around the tubules, free and in cells.

In all forms of leprosy the lymphatic glands appertaining to parts in which leprous deposit is present are characteristically affected. They are swollen and hard, and on section the gland tissue is seen to have a yellowish tinge from an infiltration which contains numerous bacilli and globi.

Albuminoid disease of the alimentary canal, liver, and spleen, and nephritis occur in a large proportion of the cases of nodular leprosy.

Diagnosis.—The touchstone in all doubtful cases is the presence or absence of anæsthesia in some skin lesion, or in some skin area. Anæsthesia is rarely absent in leprosy ; generally, in the implicated spots, it is complete, or nearly so. It should be particularly sought for towards the centre of maculæ, in the pale patches left after the fading of former maculæ, in the hands and feet, and in nodules of some standing. In no other skin disease is definite anæsthesia a symptom. Vitiligo or leucoderma—sometimes called white leprosy and by the vulgar very generally regarded as true leprosy—bears a certain resemblance to the pale post-macular patches referred to ; not to mention other features, the absence of anæsthesia in leucoderma at once settles diagnosis.

Further assistance may sometimes be got in doubtful cases from the fact that leprous spots rarely perspire. A hypodermic injection of pilocarpine is of use in bringing out this point.

The sensory and trophic lesions of syringo-myelia might be mistaken for nerve leprosy ; but the general history of the case, the history or presence of macular

eruption, of thickened nerve trunks, and of enlarged lymphatic glands in leprosy, and their absence in syringo-myelia, are mostly sufficient to establish a diagnosis.

It is hardly necessary to point out the diagnostic marks of leprosy as against syphilis, erythema multiforme, erythema nodosum, lupus vulgaris, lupus erythematosus, psoriasis, eczema, lichen planus, keloid, body-ringworm, erythrasma, pityriasis versicolor, elephantiasis arabum, etc. Mistakes can scarcely be made unless from carelessness, or by someone completely ignorant of the nature, history, and symptoms of these diseases.

In approaching the diagnosis of skin eruptions, localised pareses, muscular atrophies, and anæsthesia in patients living in, or coming from, a country in which leprosy is endemic, the possibility of their being attributable to this disease must be borne in mind. If doubt exist, and it be found feasible, search should be made in eruptions or in thickened nerves for the bacillus. If this be found the diagnosis of leprosy will be infallibly established.

Prognosis.—Complete recovery is an event so rare in leprosy that, though it may be hoped for, it must not be expected. Recovery from the actual disease itself—that is, in the sense that fresh leprous infiltration may cease to occur, and old infiltration may be absorbed, and that the bacilli may die out—is perhaps the rule in nerve leprosy ; but the effects of the leprous process are generally permanent, the trophic lesions resulting from nerve destruction being irremediable. Such cases may live, however, for many years—thirty or forty—and die of some other disease ; at best, they are but mutilated specimens of humanity.

Nodular leprosy is usually a much more acute disease than nerve leprosy, sapping the strength and general health much more effectually and more quickly. It rarely runs its full course, death being brought about by some intercurrent disease, such as, and especially, phthisis, nephritis, albuminoid degeneration of





the alimentary tract, dysentery, stenosis of the larynx, and pneumonia. It may prove fatal as a sort of galloping leprosy within a year even of its first declaring itself.

Ætiology.—*Age.*—It is open to question if leprosy has ever been seen in the fœtus. It has once or twice been reported in the newly-born. Cases are also on record of its occurrence as early as the first and second years of life ; such, however, are quite exceptional. Leprosy is extremely rare before the fifth or sixth year. In the great majority of instances the disease begins between the tenth and thirtieth year. It rarely commences after forty, although it has been known to begin up to, and even after, seventy.

Sex ; occupation ; social and hygienic conditions.—Apart from the associated social conditions as affording opportunity for contagion, sex seems to have little bearing on the liability to leprosy. The same may be said of occupation, and social and hygienic conditions in general. Very probably bad food and bad hygienic circumstances have in this, as in most germ diseases, a predisposing influence ; but they certainly cannot create a lepra bacillus and leprosy, any more than they can create an acarus and itch. This is abundantly shown by the absence of leprosy at an earlier period in countries in which, without alteration in the food or other hygienic conditions, the susceptibility of the natives to the disease was subsequently proved by its rapid spread on being introduced from without, *e.g.* the Sandwich Islands and New Caledonia ; and, also, by the disappearance of the disease, in other instances, under the influence of the segregation and isolation of lepers, without any concurrent material alteration in food or other circumstances, *e.g.* Scotland, Ireland and most European countries.

Climate.—Climate can in no way be considered a cause of leprosy, for leprosy exists in all climates and in all latitudes. But it does seem to have some

influence in determining, to a certain extent, the type the disease assumes. It would appear that the nodular form is more common in cold, damp climates; the nerve form in warm or dry climates.

The lepra bacillus.—Hansen remarks, "There is hardly anything on earth, or between it and heaven, which has not been regarded as the cause of leprosy." However true this remark may be as regards times prior to Hansen's discovery, we are now practically certain that the *lepra bacillus* is the cause of leprosy. The only gap in the evidence, otherwise conclusive, lies in our present inability to cultivate the bacillus, or to convey it by inoculation or otherwise to the lower animals, or, perhaps, to man himself.

Many attempts have been made to communicate leprosy to man by inoculation, but hitherto, with one questionable exception, all have failed. A Sandwich Islander, apparently at the time free from leprosy, was inoculated from a *lepra tubercle*. Within a month he had symptoms of leprous neuritis; two years later he was a confirmed leper; and in six years from the date of the inoculation he died of leprosy. Unfortunately the subject of the experiment was a native of a country in which leprosy was extensively endemic; and besides, he had lived among lepers—in fact, members of his family were lepers. However possible it may be that the bacillus in this instance had been communicated by the inoculation, the circumstances in which the experiment was made, and the unusual shortness of the incubation period, are against its being regarded as conclusive evidence of the inoculability of the disease.

To bridge over temporarily this important gap in the evidence, we have to fall back on the close analogy that subsists between *Bacillus lepræ* and *Bacillus tuberculosis*, the leproma and the tubercle, leprosy and tuberculosis. In consideration of this and other circumstances, it is generally conceded nowadays that *Bacillus lepræ* is the cause of leprosy, just as *Bacillus tuberculosis* is the cause of tubercle. Authorities

Van Houtem cultivated *Capnia*
bacilli in fish broth.

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differ, however, as to the way in which the bacillus is acquired.

How acquired.—It is absurd to suppose that an organism, no matter how humble its place in the scale of life, can originate *de novo*; such a hypothesis may be at once dismissed. Disregarding this, we have to consider two principal views as to the way in which the bacillus is acquired—heredity and contagion.

Heredity.—From the fact that it tends to run in families and that in certain instances it assumes the appearance of atavism, leprosy, until the *Bacillus lepræ* was discovered, was almost universally—as it still is by some—believed to be a hereditary disease. That this belief, in the same sense as that tubercle may be said to be hereditary, was well founded is quite possible; that is to say, that certain physio-pathological qualities predisposing to leprosy may be inherited. But since the discovery of the bacillus it is impossible any longer, if we properly consider it, to believe that the bacillus itself, and therefore the disease it causes, can be hereditary in the scientific sense of the word “hereditary.” Physiological peculiarities and susceptibilities may, but parasites cannot, be inherited. It is true the ovum may be infected by a germ, as in syphilis; but infection is not heredity. That the ovum can be infected at some stage of its existence by the lepra bacillus is proved if it be true that children have been born with the lesions of leprosy on them. But because leprosy is common in the descendants and blood collaterals of lepers, this is no proof of ovum infection in every, or perhaps, in any case; for family liability is quite as explicable by a hypothesis of contagion or outside infection as by a hypothesis of inherited infection. Not only may the individuals of a family inherit a family predisposition of susceptibility to the bacillus, but, as a family, the members of it are generally at one time or another closely associated, exposed to the same hygienic influences, liable to communicate by

contact with each other each other's parasites, or to acquire the parasites latent in their common surroundings. Because the members of a family simultaneously, or one after another, contract scabies, or ringworm, or typhoid, no one supposes on this account that any of these diseases is hereditary.

Without absolutely denying the possibility of ovum infection, the probability is that such an event is very rare. The age at which leprosy usually appears is against such a supposition. The latency of a germ for twenty, thirty, forty, or even seventy years is an extremely improbable thing and without parallel in pathology. Atavism, or, rather, the appearance of atavism, frequently met with in leprosy, is also against such a supposition; for, although we can understand infection of an ovum by a leper parent, we cannot understand the transmission of a germ from a grandparent to a grandchild through a parent who is not, never was, and who may never become, a leper. Such a thing would imply proliferation of the bacillus in the parent without pathological evidence of its presence.

Even admitting that leprosy is sometimes transmitted by ovum infection, this method of transmission cannot be the only one, or even a common one, for many lepers have no leper ancestors; and, as is well known, the healthy European, coming from a country in which leprosy has not been seen for generations, may acquire leprosy on visiting a country in which the disease is endemic.

If leprosy be communicated generally, or even sometimes, by parent to child by heredity, how explain the striking fact, brought out by Hansen, that of the numerous offspring of 160 Norwegian lepers who emigrated to America not one has become a leper? Or, again, the equally well attested fact that children sometimes become lepers first, their parents afterwards?

Another powerful argument against the doctrine of heredity is the circumstance that lepers become





sterile early in the disease. From this it is evident that unless the ranks of leprosy are recruited in some other way than by heredity the disease would inevitably die out in one, or at most, two generations.

From considerations such as these the view that leprosy is a hereditary disease has now few adherents among the well informed.

Contagion.—The best authorities believe that leprosy is propagated by contagion and only by contagion. The same unanimity of opinion does not obtain as to the particular way in which, or medium by which, the contagium is applied; but that it passes directly or indirectly from the infecting leper to the infected, nearly all are agreed to regard as being practically proved. The principal facts and considerations on which this important conclusion is founded are as follows:—

Leprosy is a germ disease, and therefore it cannot originate *de novo*. It must come from a pre-existing germ whose habitat may be air, soil, water, plant, beast, food, or man. That the habitat of the infecting germ is man is rendered in the highest degree probable by the fact that the germ is found in the human tissues and hitherto nowhere else; and by the fact that leprosy has never been known to appear on virgin soil independently of the prior advent of a leper. When a leper settles down in virgin country, after a time cases of the disease crop up among his companions and immediate neighbours. Some of these newly-made lepers, proceeding to a different part of the country, in time become centres for other groups of cases. Thus in the early history of the introduction of leprosy into a virgin country—as New Caledonia—the spread of the disease from individual to individual, and from place to place, can be, and has been traced.

In further proof it can be advanced that not only may a native of a non-leper country acquire the disease on visiting a leper country, but he may also communicate the disease to others, his countrymen,

on his return to his own country. There is at least one well-authenticated example of this on record. Dr. Hawtrey Benson, in 1872, showed at the Medical Society of Dublin a leper, an Irishman, who had acquired his disease in the West Indies. After his return to Ireland he slept in the same bed as his brother, who, moreover, sometimes wore the leper's clothes. In time the brother, who had never been out of the United Kingdom, became a leper and was shown to the same medical society in 1877. In this case there can be no question of fact or of diagnosis. Such a case can only be explained by contagion. Many similar, though not quite so well authenticated and conclusive, instances of the communication of leprosy by contagion are on record; but the case just mentioned is alone almost conclusive, for if leprosy is proved to be communicable by contagion in one case, the probabilities are that it is so acquired in every case.

It has been advanced against the contagiousness of leprosy, that it attacks a very small proportion only of the attendants, nurses, and doctors in leper asylums. But might not a similar objection be raised to the contagiousness of scabies or of ringworm? The conditions for successful contagion are known and can be easily avoided in the latter diseases; they are not known, and are therefore not invariably avoided in leprosy. All contagious diseases demand certain conditions for their diffusion. In some diseases these conditions are easily complied with and often concur; in other diseases they are with difficulty complied with and rarely concur. Leprosy belongs to the latter category.

Probably intimate personal contact, and certain concurrences in the phases of the disease with special conditions in the health or physiological state of the recipient—conditions which are as yet unknown, and which, for all we know, may concur only at long intervals in the intercourse of any two individuals—are necessary for the successful communication and





acquisition of leprosy. The simple implantation of the bacillus does not suffice ; for, as already pointed out, of the many inoculations that have been made only one has any claim to be regarded as having been successful.

Articles of diet—such as dried fish—have been incriminated as media for infection. The data on which it is sought to establish this and similar speculations are altogether insufficient, and do not bear investigation.

Prevention.—If it be conceded that leprosy is caused by a germ, that it is contagious directly or indirectly, and that it never breaks fresh ground unless first introduced from without and by a leper, then the leper must be regarded as a source of danger, and, *quâ* leprosy, the only source of danger to any community he may live amongst ; and a sure, and the most effectual way of suppressing the disease is the thorough isolation of existing lepers. There are many difficulties, however, especially in such countries as India, in giving practical expression to what appears to be a perfectly logical conclusion—difficulties springing from the rights of the individual, finance difficulties, difficulties arising from concealment or incorrect diagnosis, as well as from the continued introduction of fresh cases from without. These and other obvious obstacles, incident to any attempt at a wholesale system of thorough isolation, are so great that the most that can be hoped for at the present time, and in the present state of public opinion, is some modified system of segregation and isolation, such as has worked so successfully in recent years in Norway.

Where possible, therefore, lepers should be segregated in isolated asylums which should be so conducted as to prove attractive. Those who cannot be made, or persuaded, to enter these asylums should be isolated as much as possible from their families and the public ; scrupulous cleanliness of their persons and houses should also be insisted on. Lepers ought not to be allowed to beg in the streets as is often the

case in Eastern cities, to keep shops, or to handle food or clothes intended for sale, to wander about the country as pedlars or mendicants, to hire themselves out as servants or prostitutes, or to frequent fairs and public places. All lepers in the ulcerative stage of the disease, when it is to be presumed that myriads of bacilli are being constantly given off from their sores, should be still more scrupulously isolated, their discharges, clothes, and dressings being systematically destroyed or disinfected. A child born of a leper should at once be removed from the diseased parent, and, if necessary, cared for at the public expense.

If laws were enacted to give effect to such common-sense precautions, and if these laws were faithfully carried out, the best results might be looked for. Leprosy is feebly contagious, or rather, the conditions for successful contagion rarely recur; so rarely, that it is more than probable that under such a modified system of segregation and isolation as that indicated, they would recur so seldom that the disease would rapidly die out.

Vaccination.—It has not been actually proved that leprosy can be communicated by vaccination, although there is some evidence in favour of such a supposition. But, although this has not been proved, it is an obvious and very desirable precaution, in countries in which the disease is endemic, to take care that the vaccinifer is not only not the subject of actual leprosy eruption, but, also, that he or she comes from a family and community free from leprosy. An apparently healthy vaccinifer may contain lepra bacilli in a latent state, be, in fact, a potential leper capable of communicating the disease.

Treatment.—Scrupulous and systematic attention to personal and domestic hygiene and cleanliness; frequent bathing and the free use of soap; frequent changes of underclothing; good food; fresh air; light work; the avoidance of over-strain, fatigue, and of exposure to bad weather; these things are all of prime importance in the treatment of leprosy,





and should be insisted on. It has been found that most lepers on being placed in favourable hygienic conditions improve, at all events for a time, and that in a small proportion the disease by these means may sometimes be actually arrested. Europeans who have contracted leprosy in the tropics, almost invariably undergo temporary improvement on return to the more bracing climate and more nutritious diet of their native lands.

Many drugs have been regarded, from time to time, as being more or less of the nature of specifics in the treatment of leprosy. But, though some of these drugs appear for a time to do good, and in consequence acquire a certain degree of popularity, hitherto all of them, one after another, have sooner or later fallen into disfavour. There is no good reason to think that any known medicinal substance is really a specific for leprosy, in anything approaching the sense that mercury and iodide of potassium are specific in syphilis. One is very apt to be deceived in estimating the value of a drug in leprosy. The leper applies for treatment generally during, or soon after, one of the periodical exacerbations of the disease, and when the nodules and other eruptions are active and well pronounced. In the natural course of events, and without treatment of any description, especially if the patient be placed under favourable hygienic conditions, these acute manifestations tend to become quiescent, and the disease temporarily to ameliorate. Observers are too apt to attribute this natural and temporary amelioration to whatever drug the patient may happen to be taking at the time. Moreover, in judging of the value of any drug in leprosy, it must be remembered that the disease may be arrested spontaneously, or even be recovered from, and that without the use of any drug whatever.

Chaulmoogra oil, in doses of from two to ten up to forty drops or more, according to tolerance, three times a day, together with inunction of the same

drug mixed with some oil, is a favourite remedy with English practitioners. Such lepers as can assimilate large doses of this drug appear to derive benefit. Sandwith has reported (*Journ. of Tropical Medicine*, 1900) a case in which marked benefit followed persistent hypodermic administration of chaulmoogra. I have a leper at present under my care who, in addition to 70 minims of chaulmoogra three times a day by the mouth, receives a hypodermic injection daily of one drachm. The improvement in this case is very marked. The bacilli, however, are just as abundant in the nodules as before treatment began, and there is no alteration in the appearance of the bacilli. Gurjun oil, once in favour, seems to have been abandoned.

Unna claims to have cured several cases by the internal administration of ichthyol in increasing doses, combining the internal medication with vigorous rubbing of the arms and legs twice a day with pyrogallie acid (10 per cent.) in lanolin, and the cheeks and trunk with chrysophanic acid (10 per cent.) in lanolin; at the same time applying to the forehead and chin a plaster of chrysophanic and salicylic acids with creasote, changing the plaster every day. The treatment is continued for a month, and is then followed by a course of warm baths before being resumed.

Tuberculin has also been tried. It produces a local and a general reaction, which is sometimes curiously delayed for one or two days. So far from doing good it seems to aggravate the disease, causing fresh eruptions, and, also, causing bacilli to appear in the blood.

Dr. Radcliffe Crocker has recorded (*Lancet*, August 8th, 1896) two cases of leprosy in which great improvement followed weekly hypodermic injections of one-fifth of a grain of perchloride of mercury.

Iodide of potassium aggravates leprosy if given in full doses; it not only affects the general health prejudicially, but it causes fresh eruptions to appear.





Danielssen regards salicylate of soda, combined with cod-liver oil, quinine and iron, good food, and good hygiene, as the best treatment for leprosy. He claims for the salicylate, if commenced within the first few months from the appearance of the disease, that it sometimes effects a cure. He begins with fifteen grains, four times a day, and gradually increases the dose during six months or a year.

I have lately tried thyroidin in a case of nerve leprosy. The patient has improved marvellously during the three years he has taken it.

Hydroxylamin, euophen, naphthol, salol, methylene blue, and aristol have also been tried recently; the results have not proved encouraging.

Nerve stretching, with or without nerve splitting, has been strongly advocated (McLeod) for the cure of leprous neuralgia, anæsthesia, muscular atrophy, and other trophic lesions. At the best they can benefit the local lesion alone, and that but temporarily, and only in the earlier stages of the leprous neuritis before the nerve has undergone fibrous transformation.

In the case of leprous nodules spreading on to the cornea and threatening to interfere with the line of vision, Broeckmann has shown that the extension of the leproma may be arrested by division of the cornea on the pupillary side of the lesion; it is found that the bacilli do not traverse the cicatrix. Tarso-rhaphy for ectropion of the lower lid; iridectomy for iritis, or synechiæ; tracheotomy for laryngeal stenosis; and necrotomy for bone disease, may sometimes have to be performed. Horder strongly recommends amputation for perforating or other forms of ulceration; the general health is much improved by the removal of such sources of sepsis. The existence of leprosy does not materially interfere with the success of surgical operations. I once removed an enormous elephantiasis of the scrotum from a confirmed leper; the presence of the leprosy did not prevent sound healing of the extensive operation wound, the man

making a good recovery so far as the operation was concerned.

If only one tubercle, or one limited lepra macula is present, and there have been no constitutional signs of a general invasion, it is advisable to excise freely the affected spot. It is just possible that in this spot we may be dealing with the primary lesion of leprosy—the point of invasion—and that as yet the disease is limited to this. At all events, to excise this can do no harm; and in face of the terrible fate which is otherwise in store for the victim of this infection, it would be wrong to withhold any possible chance of escape, however small, such a trifling operation might afford. A case has been recorded (*Arch. de Méd. Expér.*, 1st Jan., 1895) in which a child, twenty-seven months old, presented on one temple a minute, red, anæsthetic spot which, on microscopic examination, proved to contain lepra bacilli. The child's brother was a confirmed leper. Six months after the discovery of the spot it was excised and the wound cauterised. This was done in October, 1893; in April, 1894, the child showed no signs of leprosy.

As already mentioned, the occurrence of acute disease may cause absorption of leprous deposit. This has been more particularly observed after erysipelas. It has accordingly been proposed to treat leprosy by inoculations of the erysipelas streptococcus, or by injections of the filtered toxin obtained from cultures of that organism (Impey).





CHAPTER XXIX.

YAWS (FRAMBÆSIA).

Definition.—Yaws is a contagious, inoculable disease characterised by an indefinite incubation period followed usually by fever, by rheumatic-like pains, and by the appearance of papules on the skin, which generally develop into a fungating, granulomatous, encrusted eruption. It runs a chronic course; is mostly protective against a second attack; and, to a certain extent, is influenced by mercury and potassium iodide.

Geographical distribution.—Yaws is widely diffused throughout the greater part of the tropical world. In certain places it is very common—as in tropical Africa, particularly on the west coast; in many of the West India islands; in Ceylon, where it is one of perhaps several diseases included under the term *parangi*; in Fiji, where it is known as *coko*; in Java; in Samoa; and in many of the islands of the South Pacific. It is difficult to say to what extent it exists in India; some deny its presence there altogether, but recent observations show that it does occur there to a limited extent. Powell (*Ind. Med. Gaz.*, Sept., 1894, the *Brit. Journ. of Dermat.*, No. 98, Dec., 1896) has recognised and described it as occurring in Assam. Barker, of Borneo, has recently shown that the skin disease of the Eastern peninsula, described by Brown (*Brit. Journ. of Dermat.*, No. 56, vol. v.), under the name of *purru*, as being common in parts of the Malay peninsula, is yaws. If yaws occurs in China, it is certainly rare there—at all events on the coast. In some of the West India islands, and in Fiji, almost every child passes through an attack. In the latter, according to Daniels, those children who do not

acquire the disease in the ordinary way are inoculated with it by their parents, who regard an attack of yaws as an occurrence more or less necessary and wholesome. Nicholls (*Report on Yaws*, Blue Book, 1894) has made a careful and admirable study of West Indian yaws. His inclination is to look upon parangi, coko, and similar Asiatic and Pacific island diseases as specifically different from the African and West Indian disease. Daniels, however—a most accurate observer, who has had extensive experience both in Fiji and in British Guiana—shows very clearly that in both places the diseases are identical. Probably the view that certain forms of the parangi of Ceylon are not yaws is likewise incorrect.

It is impossible at the present day to settle the point, but it seems probable that yaws was originally an African disease, and, so far as America and the West Indies are concerned, that it was introduced by negro slaves. In the days of West Indian slavery the specific and infectious nature of yaws was thoroughly recognised. The planters from commercial, apart from other, considerations, by instituting yaws houses and other repressive measures, took much trouble to keep the disease under. Since emancipation has permitted the West Indian negro to revert to some extent to the state of savagery from which he had partly emerged, yaws has again become very prevalent, and is now a principal and loathsome feature in the morbidity of these islands.

Symptoms.—*The initial fever.*—In yaws there is an incubation stage of very variable duration—two weeks to six months*—the appearance of the characteristic eruptions being preceded by a certain amount of constitutional disturbance. The intensity of the general symptoms varies within wide limits. Sometimes they are hardly perceptible, and are not

* Paulet, who inoculated fourteen healthy persons with yaws, found the first lesion in from twelve to twenty days; Charlotis, in twenty-eight inoculations observed a papule at the site of inoculation after fourteen days. Naturally acquired yaws is reputed to have a longer incubation period than the inoculated disease.





complained of; usually there is well-marked malaise with rheumatic pains. Occasionally there is severe constitutional disturbance, lasting for about a week, with rigor, smart fever— 100° to 103° —persistent headache, pains—worse at night—in the long bones, joints and loins, and sometimes gastric disturbance and diarrhœa. During the decline of these constitutional symptoms the eruption appears.

Stage of furfuraceous desquamation.—The skin becomes harsh and dry, loses its natural gloss, and here and there patches of light-coloured, very fine furfuraceous desquamation, best appreciated with the aid of a lens, are formed. These patches are mostly small and circular; occasionally they are oval, irregular, or form rings encircling islets of healthy skin. Their extent and number are very uncertain. They are scattered irregularly over limbs and trunk; occasionally they may be almost confluent, the patches coalescing and giving rise to an appearance as if the entire skin had been dusted over with flour. On the other hand, this furfuraceous desquamation may be so slight as to be overlooked. In other instances it may be very marked; the heaping up of desquamating epidermic scales producing white marks, very evident on the dark skin of a negro or Oriental.

This patchy, furfuraceous condition of the skin occurs not only at the early stages of yaws, but it may persist throughout the attack, or reappear as a fresh eruption at any period of the disease.

The yaw (Fig. 48).—When the furfuraceous patches have been in existence for a few days minute papules appear in them. Describing these papules, Nicholls remarks that, in examining them with a lens, “they are seen to be apparently pushed up from the rete malpighii through the horny epidermis, which breaks over their summits and splits in radiating lines from the centre, the necrosed segments curling away from the increasing papule. When the papules become about a millimetre in height and breadth, a yellow point may be observed on the

summits . . . consisting not of a drop of pus under the epidermis . . . but of a naked, cheesy-looking substance, which cannot be wiped away unless undue force be used. Frequently a hair will be observed



Fig. 48.—Yaws.

issuing from this yellow substance, thereby indicating that the hair follicles are the centres of the change going on." This eruption may persist during the entire attack, or it may appear at any time during the course of the disease. When extensive and occurring late, it indicates a protracted attack.

The papule, having arrived at this stage, may either cease to grow, the apex becoming depressed,





cupped, and lined with the yellow cheesy material alluded to ; or it may go on, increasing in size, to the formation of the typical yaw. In the latter case the lesion gradually grows into a rounded excrescence, the yellow material at the top widening out so as to form a complete cap encrusting the little tumour. The yaw so formed may be no larger than a split pea ; or it may attain the breadth of a crown piece. The smaller tumours are hemispherical ; the larger are more flattened or even depressed at the centre, possessing everted, somewhat overhanging, rounded edges. Occasionally, though rarely, a big yaw may include an area of sound skin. Several yaws may coalesce, and together cover a large and irregular surface, as an entire cheek, a popliteal space, or the dorsum of a foot. In the case of these large yaws, the surface of the growth is apt to be irregular and fissured. The neighbourhood of the mouth and anus are favourite sites for coalescent yaws ; in such situations the moisture of the parts softens and removes the crust wholly or in part, so that the surface, in addition to being fissured, may be more or less bare, sodden, and fungoid.

The crust which caps and encloses an uninjured yaw is yellowish, granular, blotched with blood stains and encrusted dirt. At first the crust is somewhat moist, but gradually it becomes dry, brown, and black even. The crusts are firmly adherent, requiring some force to remove them ; a proceeding which, though painless, may entail a little oozing of blood. Deprived of its crust the little swelling is seen to be red in colour, and generally smooth and rounded on the surface. According to size, it stands out anything from one-eighth to six-eighths of an inch above the surrounding healthy skin. Immediately after removal of the crust the exposed surface begins to pour out a pale, yellowish-grey, viscid fluid, which soon becomes inspissated, rapidly forming a fresh cap to the yaw. Pus, unless as a consequence of irritation, is not, as a rule, found under the crust.

Although the formation of the papules and yaws is attended with much itching, the yaw itself is not at all sensitive; the tumour may be touched with acid even with impunity—a diagnostic point of some importance.

The yaw usually attains its maximum development in two weeks; for several weeks longer it remains stationary before beginning to shrink. The crust then thins, shrinks, darkens, separates at the periphery and at last falls off, disclosing at the site of the former fungating mass a slightly thickened (pale—Nicholls, hyperpigmented—Daniels) spot of fairly sound skin.

Ulceration. Such is the normal process of evolution and involution of a yaw. But it sometimes happens that the tumours, in place of becoming absorbed, break down and ulcerate, the ulceration, however, being confined to the yaw itself. In other instances ulceration goes deeper and extends circumferentially, giving rise to extensive sores with subsequent cicatricial contractions. Such ulcerations may or may not be encrusted. With the development of the deeper and wider forms of ulceration, the typical lesions of yaws may disappear for a time, or perhaps permanently. In the latter case the ulcers are said not to be infective, and do not communicate yaws; they are, therefore, to be regarded rather as complications or, it may be, sequelæ. Such ulcers may persist for years. Ulceration, according to Nicholls, occurs in about 8 per cent. of cases.

Onychia.—Yaws may occur around or under a nail and give rise to a troublesome form of onychia.

Foot yaws.—When a yaw develops on the sole of a foot, in consequence of being bound down by the dense and thick epidermis it causes much suffering. Spreading laterally under the thick, leathery, and unyielding epidermis, it may attain a large size. After a time the epidermis over the growth gives way, splitting in a radiating fashion. Pressure being thus removed the yaw fungates, and suffering diminishes.



A cracked scaly condition of the hands and feet, sometimes persisting for years, is not unusual in negroes, and must not be confounded with yaws, although not infrequently the two conditions co-exist.*

Distribution.—The yaws may be scattered over the whole body; or the crop may be limited to one or two growths; or they may be confined to a circumscribed region of the skin. They are commonest on exposed parts, on the anterior surface of the body, and on parts especially liable to injury, as the feet and legs. They are most frequently found on the lower extremities; rarely on the scalp, and still more rarely in the axillæ. They are hardly ever seen on mucous surfaces unless about the lips, around the angles of the mouth, and in the nostrils, where they often form clusters.

Duration and recurrences.—Yaws lasts for weeks, months or years, its duration depending on the general health, idiosyncrasy, hygienic conditions, and the treatment employed. Mild cases in healthy subjects finish in about six weeks. In other instances, especially in the debilitated, the disease runs on for months, successive crops of eruption being evolved. Sometimes these recurrences may stop short at the

* In the course of time the West Indian negroes have adopted a peculiar jargon—a mixture of French, English, and Spanish—to designate the various manifestations of yaws. The scaly patches are known in some of the islands as “pian dartres,” in Jamaica as “yaws cacca”; the papular stage of eruption as “pian gratelle”; when the papular eruption occurs as a late symptom, it may be called “pian charaib,” or “guinea-corn yaws.” The developed yaw is sometimes known as “bouton pian.” “Tubboes,” “tubba,” “crabs,” “crappox,” “crabes,” are expressions applied to the painful manifestations on the soles of the feet. Forms of chronic dermatitis on hands and feet are called “dartres,” “tubhoe,” “crabs,” “dry tubboes”; or where exudation goes on between cracks in soles or palms, “running crab yaws.” A large persistent yaw is sometimes known as the “mother,” or “grandmother,” or “mama-pian”; smaller yaws as “daughter” or “grand-daughter” yaws. Yaws which show themselves some time after the disease appears to have subsided are called “memba” (remember) yaws. Yaws coalescing in the form of a ring are called “ringworm” yaws.

stage of desquamation, or at the papular stage, or they may proceed to the formation of typical yaws. The recurrences are usually preceded by feverishness, pains in the bones and joints; and the successive crops may either be limited and partial in their distribution, or they may be general. In Fiji, Daniels states, the average duration of an attack of yaws is about one year.

The general health.—With the exception of during the initial fever, or during one of the recurring febrile relapses, the general health is not as a rule affected. Occasionally there is debility and cachexia; or there may be enlargement and tenderness of the lymphatic glands. In other instances the rheumatic pains are a principal feature and may be very severe.

Persistent yaws.—That yaws sometimes effects a permanent hold is shown by the persistency with which they occasionally continue to recur during many years. In such cases the lesion has always the characters of a true yaw, and cannot be regarded as a “secondary” or “tertiary” manifestation in the sense in which these terms are applied to the late lesions of syphilis.

Question of sequelæ.—Mention is often made of nodes, of gummatous-like thickenings, and of punched out and serpiginous ulcerations in connection with yaws. Most recent authorities regard all such phenomena as being generally the results of an independent, though concurrent, syphilitic infection.

It sometimes happens that an individual who, years previously, passed through an attack of yaws is affected with destructive ulceration of the soft palate, or of the mucous membrane and cartilage of the nose. Opinions differ as to the true relationship between this special type of ulceration and the antecedent yaws; some holding that it is a later manifestation of yaws, others regarding it as purely the result of an independent syphilitic taint. As regards the West Indies the question is one which,





considering the great frequency of both acquired and congenital syphilis there, it is hard to decide. Daniels says that in Fiji, where syphilis is unknown among the natives, these destructive ulcerations of palate and nose, together with a skin affection like lupus vulgaris—all of which he says are amenable to potassium iodide—are not uncommon; he is inclined, therefore, to regard them as true sequelæ of yaws. Corney appears to entertain a similar opinion, for in his annual statistical return of the Colonial Hospital, Fiji, 1896, he classifies eleven cases under the heading “Lupoid ulceration of posterior nares and adjoining pharyngeal parts (tertiary frambœsia).” Powell (*Ind. Med. Gaz.*, Aug., 1898) describes two cases, mother and child, in whom, concurrently, a uniform swelling of the proximal phalanges of both hands occurred during the third year of an attack of yaws. The bones to the touch gave the impression of being rarefied. The swellings yielded to mercury.

Question of a primary sore.—Another point in the symptomatology of yaws is the question of the occurrence of a primary sore, as in syphilis. Numa Rat says there is such a sore, but that it is usually overlooked. He describes it as a papule with a pale yellow material at its apex, which may remain a papule, or which, after seven days, may ulcerate and subsequently cicatrise. Other observers do not agree with this. They say that though yaws virus applied to a pre-existing ulcer may render it unhealthy-looking and cause it to fungate like an ordinary yaw, yet successful puncture inoculations, although they sometimes give rise to a yaw at the point of inoculation, do not by any means always produce a local lesion, much less an ulcer. As the lower animals appear not to be susceptible to yaws, this and other interesting points cannot be settled by experiment on them.

Mortality.—Although in the literature of the subject reference is made to deaths from yaws, yet, judging from the statistics collected by Nicholls, the mortality must be very small indeed. In 7,157 West

Indian cases, treated in various yaws hospitals, there were only 185 deaths—a mortality of 25·8 per thousand; a death-rate, as Nicholls points out, less than the average annual death-rate in one of the islands—Antigua. Doubtless although yaws itself rarely directly proves fatal, intercurrent diseases, such as sloughing phagedæna and phagedænic ulceration, predisposed to by the skin lesions, occasionally do so. If the verruga of Peru be a form of yaws, then, under certain conditions, yaws becomes a very dangerous disease.

Ætiology.—*Contagion and heredity.*—As yaws is highly contagious, all circumstances favouring contact with the subject of the disease favour its occurrence. Simple skin contact does not suffice for infection; a breach of surface is necessary. Probably the virus is often conveyed by insect bites, or by insects acting as go-betweens and carrying it from a yaw sore to an ordinary ulcer. Thus the disease often commences in a pre-existing ulcer. Cases are prone to originate in certain dirty houses, the virus from previous yaws patients seemingly impregnating the floors and walls of the filthy huts in which the latter had resided. In this way the disease may be acquired without direct transference from an existing case.

Yaws is neither hereditary nor congenital. A pregnant mother suffering from yaws does not give birth to a child suffering from the same disease; or one which will subsequently develop yaws, unless the virus be first introduced directly through a breach of surface after birth. It is not conveyed by the milk; nor does a suckling suffering from yaws necessarily infect its nurse.

Age, sex, occupation, race.—Although two-thirds of the cases in the West Indies occur before puberty, no age is exempt. Three males are infected to every female attacked. Occupation has no manifest influence. In the West Indies, Europeans, Chinese, and Indians catch the disease if exposed to the contagion.



Morbid anatomy and pathology.—No visceral changes have been found in yaws, although, of course, when yaws concurs with syphilis, gummata, etc., may be found; but in this case the concurrent gummata belong to the syphilitic and not to the yaws infection.

The tumours on the skin are granulomata made up of round or spindle-shaped cells, held together by a small amount of connective tissue and abundant blood-vessels. The focus of the circumscribed cell proliferation is the papilla which becomes very much swollen, and the malpighian layer. The cause of the proliferation is probably some micro-organism; what this may be has not been satisfactorily determined. Both Pierez and Nicholls found a micrococcus in great profusion in the growth and exudation. This they succeeded in cultivating; but the causal relationship of the bacterium to the disease has not been established, inoculations of cultures into the lower animals proving negative.

Diagnosis.—A painless, insensitive, larger or smaller, circular, encrusted, red granulosomatous excrescence occurring in the endemic district, is almost certainly yaws. The most important point in connection with yaws, both as regards diagnosis and ætiology, is its relationship to syphilis. It has been, and is still, held by some distinguished authorities, Hutchinson for example, that yaws is possibly syphilis modified by race and climate; and certain features which the two diseases have in common are pointed to in support of the contention. The discussion is bound to continue until the respective germs of yaws and syphilis have been separated, cultivated and inoculated. So far as clinical and microscopical evidence goes, it is decidedly in favour of, not to say conclusive for, regarding the two diseases as specifically distinct. There are many points of contrast in their clinical features. I may mention the primary sore, the infection of the foetus, the adenitis, the exanthem, the alopecia, the absence

of itching, the iritis, the affection of the permanent teeth, the bone and eye affections, the congenital lesions, the polymorphism of the eruptions, the nerve lesions and the gummata of syphilis. All these are wanting in yaws. Moreover, both diseases may concur in the same individual (Powell cites two cases, and Charlouis two, of syphilis supervening on yaws); and antecedent syphilis certainly does not confer immunity as against yaws. Yaws may die out in a community, as in British Guiana (Daniels, *Brit. Journ. of Dermat.*, November, 1896), yet syphilis remain; yaws may be universal in a community, as in Fiji, and yet true syphilis, whether as an acquired or congenital disease, be unknown. Finally, syphilis has never been shown to give rise to yaws, nor yaws to syphilis; neither, so far as known, has yaws been evolved in any community from syphilis, or appeared independently where the possibility of its having been introduced from a recognised yaws centre could be excluded with certainty.

The therapeutical argument for the identity of the two diseases is a very fallacious one. Sulphur will cure scabies and pityriasis versicolor; yet from this circumstance we may not conclude that these diseases are identical. The same may be said in respect of the influence of mercury and iodine on syphilis and on yaws.

Prophylaxis.—This resolves itself into the adoption of measures to prevent contagion. These are, the isolation and segregation of the affected; the dressing and treating of wounds in the hitherto unaffected; the application of antiseptic ointments to yaws sores so as to obviate the diffusion of germs; the purifying or destruction by fire of houses or huts notoriously infected; the prevention of pollution of bathing water by yaws discharges.

Treatment.—All are agreed as to the propriety of endeavouring by good food, tonics and occasional aperients to improve the general health. Most are agreed as to the propriety of endeavouring to procure





a copious eruption by stimulating the functions of the skin by warm demulcent drinks ; by a daily warm bath with plenty of soap ; and, during the outcoming of the eruption, by such diaphoretics as liquor ammoniæ acetatis, guaiacum, etc. Confection of sulphur is also recommended as a suitable aperient ; it may be taken frequently in the early stages of the disease. All are agreed as to the propriety of avoiding everything—such as chill—tending to repress the eruption ; warm clothing is therefore indicated. Many use mercury or potassium iodide, or both, after the eruption is fully developed. These drugs have undoubtedly the power of causing the eruption in yaws to resolve. Some practitioners rarely use them or, if they use them, do so only at the latest stages of the disease, considering that relapses are more prone to occur after their too early employment. Mercury, owing to its proneness to cause anæmia, is less frequently employed than potassium iodide. Where the eruption is persistently squamous, or papular, arsenic is frequently prescribed. Some touch the yaws with sulphate of copper ; some apply nitrate of mercury ointment ; others iodoform ointment ; others leave them alone, confining their local measures to the enforcement of cleanliness. When the soles of the feet are attacked, the feet ought to be soaked in warm water to soften the epidermis, which should then be cut away sufficiently to liberate the subjacent yaw. Ulceration must be treated on ordinary principles. During convalescence, iron, arsenic, and quinine are indicated.

CHAPTER XXX.

VERRUGA PERUANA.

IN certain narrow valleys of the Andes, between the ninth and sixteenth parallels of south latitude, and at an elevation of from 3,000 to 10,000 feet, an aggravated form of a disease in some respects closely resembling yaws, and locally known as "verruca," is endemic.

The peculiar initial rheumatic-like pains and fever are apparently the same in character as those of yaws, only more severe. Just as in yaws, one attack of verruga confers practical immunity. The constitutional symptoms likewise subside on the appearance of the skin lesion which, judging by the published descriptions, is a granuloma macroscopically and microscopically identical with that of yaws. Just as in the latter disease, the eruption may be sparse or abundant, discrete or confluent. As in yaws, individual granulomata may fail to erupt; others may subside rapidly; others again may continue to increase, and then, after remaining stationary for a time, gradually wither, shrink, and drop off without leaving a scar. If difference there be in their clinical features between verruga and yaws, apparently it is more one of degree than of kind.

In verruga the initial fever may continue for weeks, or even for months. It is very severe in many instances. Often it exhibits features like those of a malarial infection, including intermittency, profound anæmia, and sometimes enlargement of the spleen and liver. Very probably in such cases it is the outcome of a compound infection—verruca attacking a malarial subject. Not infrequently in the endemic



district a certain type of fever, believed to be verruga fever, proves fatal before the appearance of definite skin manifestations such as would justify a positive diagnosis. This was apparently the case with a medical student, named Carrion, who inoculated himself with blood from a verruga granuloma. The symptoms in his case closely resembled those of the very deadly fever referred to, which is known locally as Oroya fever.

In addition to the severity of the fever and rheumatic pains, the Peruvian disease is remarkable for the tendency to spontaneous hæmorrhage exhibited by the skin lesions. Apparently this peculiarity is attributable, like the hæmorrhages in the affection known as "mountain fever," to the diminished atmospheric pressure at great altitudes; for when the patients descend to the lower valleys, or to the sea level, the tendency to bleeding ceases. Possibly, the unusual vascularity of the swellings, which are sometimes permeated by a network of cavernous sinuses, also arises from the same circumstance.

In yaws we find no mention made of the occurrence of fungating granulomata in any situation but the skin. In verruga it would seem that the tumours may develop on mucous surfaces—in the œsophagus, the stomach, intestine, bladder, uterus, and vagina. Hence the dysphagia—a common symptom—and the occasional occurrence of hæmatemesis, melæna, hæmaturia, and bleeding from the vagina in the last-named disease.

The geographical distribution of verruga, so far as known, is singularly limited; it is confined to certain valleys, the inhabitants of neighbouring places being exempt. It is said that the disease may be acquired in merely passing through the endemic districts; and that, unlike yaws, the domestic animals in these districts, as well as the human inhabitants, are subject to the disease.*

* Two elaborate memoirs on this disease have recently appeared "*La Maladie de Carrion*," and an article in *La*

Treatment.—It appears that cold tends to repress the development of the eruption, and that until this appears fever and pain persist. For this reason as well as to avoid the hæmorrhage from the lesions when they do erupt, the patients should quit the heights and descend to near the sea level. External hæmorrhages, when they do occur, must be treated by graduated pressure; otherwise, the local as well as the general treatment is the same as for yaws.

Presse Médicale (July 27th, 1898), both by Dr. M. E. Andinzola. It is curious that, although animals are said to be subject to the disease, Carrion chose to experiment on himself. No mention is made in the works referred to of experiments on animals, although Carrion is lauded for his enthusiastic devotion. The reader might consult an article in Pasteur's "*Annales*" (Sept. 25th, 1898), "*Note sur la Bacteriologie de Verruge du Pérou*," by M. Charles Nicolle.



CHAPTER XXXI.

ULCERATING GRANULOMA OF THE PUDENDA.

Geographical distribution.—Drs. Neal, Ozzard, Conyers and Daniels (*Brit. Guiana Med. Ann.*, 1896) describe a peculiar form of ulcerating granuloma affecting the pudenda in dark-skinned races. Their observations were made in British Guiana and, principally, on West Indian negroes. Daniels believes that he has seen a similar or the same disease in Fijians. The “serpiginous ulceration of the genitals” referred to by Macleod (*Ind. Med. Gaz.*, 1882) and recently by other writers in India, more especially by Maitland (*Ind. Med. Gaz.*, 1899), is undoubtedly the same disease, examples of which I can recollect having seen in South China. Taylor, of New York, has seen it in whites in the United States, and Goldsmith (*Journ. of Trop. Med.*, 1899) has also met with it in aborigines in North Australia. Doubtless, therefore, although hitherto little notice has been taken of it, this disease is widely distributed in the tropics.

Age and sex.—This form of ulceration has not been observed before puberty; it has been found only after thirteen or fourteen and up to forty or fifty. It occurs in both sexes, but particularly in women.

Symptoms.—The disease commences in the great majority of cases somewhere on the genitals or groins as an insignificant, circumscribed, nodular thickening and elevation of the skin. The affected area, being covered with a very delicate, pinkish, easily-rubbed-off epithelium, excoriates readily, exposing a surface prone to bleed and break down, although rarely ulcerating deeply. The disease advances in two ways; by continuous eccentric peripheral extension and by auto-infection of an opposing surface.

In its extension it exhibits a predilection for warm and moist surfaces; particularly the folds between the scrotum and thighs, the labia, and the flexures of the thighs (Fig. 49). Its extension is very slow, years elapsing before it covers a large area. Concurrently with peripheral extension a dense, contracting, uneven, readily-breaking-down scar forms

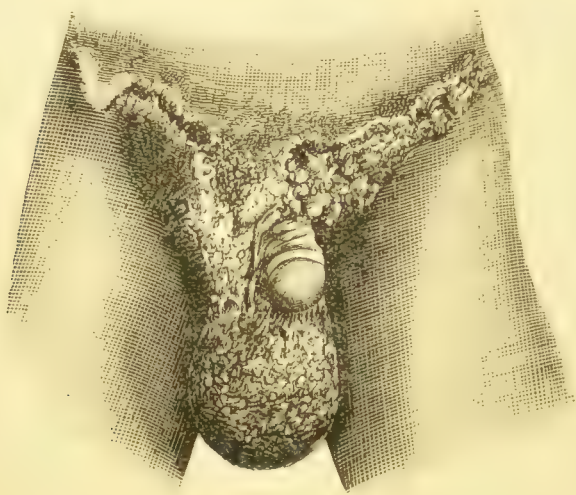


Fig. 49.—Ulcerating granuloma of the pudenda in the male.

on the surface travelled over by the coarsely or finely nodulated active new-growth which constitutes the peripheral part of the diseased area. Occasionally islands of active disease spring up in this scar tissue; but it is at the margin of the implicated patch that the special features of the affection are to be observed. In a case of some standing there is found a large area of white or irregularly pigmented, perhaps excoriated, unsound, contracting, folded, and dense cicatrix; surrounded by a narrow, serpiginous, irregular border of nodulated, somewhat raised, red, glazed, delicately skinned or pinkish, superficially ulcerated or cracked new-growth.

In the case of the female (Fig. 50) the disease may





extend into the vagina, over the labia, and along the flexures of the thighs. In the male it may spread over the penis, involve the glans, scrotum, and upper part of the thighs. In either sex it may spread in the course of years to the pubes, over the perineum, and as far back as the region of the coccyx. At times a profuse watery discharge exudes and even



Fig. 50.—Ulcerating granuloma of the pudenda in the female.

drips from the surface of the new-growth, soiling the clothes, soddening the skin, and emitting a peculiarly offensive odour. In this condition the disease, slowly extending, continues for years, giving rise to inconvenience and perhaps seriously implicating the urethra, vagina, or anus, but not otherwise materially impairing the health.

Histology.—On microscopical examination the new growth at the margins of the sore is found to be made up of nodules, or masses of nodules, consisting of round cells having large and, usually, badly-staining nuclei. These cells are embedded in a delicate fibrous reticulum. The nodular masses are,

for the most part, covered by epithelium, their under surfaces merging gradually into a dense fibrous stroma in which small clusters of similar round cells are here and there embedded. The growths, though very vascular, contain no hæmorrhages; and there are no signs of suppuration or of caseation, no giant cells, and no tubercle bacilli. In vertical section of the small nodules the round cell mass will be found to be wedge-shaped, the base of the wedge being towards the skin, the deep-lying apex usually pierced by a hair or two. The growth is found around sebaceous follicles, blood-vessels, lymphatics, and sudoriparous glands; but it is specially abundant, and most deeply situated, around the hair follicles. No characteristic micro-organism has hitherto been discovered. For a detailed description of the histology the reader should consult an elaborate article by Dr. James Galloway, in the *British Journal of Dermatology*, April, 1897.

Ætiology.—There is reason for believing that the disease is often, if not generally, a venereal one. Maitland has seen it in the mouths of a husband and wife. He considers it may be inoculated on other forms of venereal sore, such as an ulcerating bubo, and that a compound sore may result. The nature of the virus is unknown.

Diagnosis.—In British Guiana, as elsewhere, malignant and syphilitic ulcerations of the groin are common enough; the disease under notice, however, differs widely from these clinically, histologically, and therapeutically. It is characterised by extreme chronicity—ten or more years—by absence of cachexia or of any tendency to cause death, by non-implication of the lymphatic system, and by non-amenability to mercury and iodide of potassium. The disease which it most resembles is lupus vulgaris. From this it differs inasmuch as it is practically confined to the pudendal region, affects mucous as well as cutaneous surfaces, tends to follow in its extension the folds of the skin, is not associated with the tubercle bacillus, giant cells, caseation, or other evidences of tuberculous disease.





Treatment.—This disease is very rebellious to treatment. Scraping and caustics, including the actual cautery, have been freely employed ; but, although some improvement may be effected by these means, new nodules almost invariably spring up in the resulting cicatrix. Complete excision, where practicable, offers the best chance of permanent cure ; of course, such a proceeding must be undertaken before large areas and important passages have become involved. Conyers and Daniels found that camphor and carbolic acid in equal parts, and salicylic acid in unguentum creasoti, thirty to forty grains to the ounce, give the best results ; although in no instance have they succeeded in effecting a complete cure, the growth invariably recurring in the scars soon after discontinuance of treatment. Maitland recommends the repeated application of Vienna paste followed by boric acid poultices. Mercury and iodide of potassium are useless.

CHAPTER XXXII.

ORIENTAL SORE.

Definition.—A specific ulcerating granuloma of the skin, endemic within certain limited areas in many warm countries. It is characterised by an initial papule which, after scaling and crusting over, breaks down into a slowly extending and very indolent ulcer. Healing after many months, it leaves a depressed scar. The sore is inoculable and, to a considerable degree, protective against recurrence.

Geographical and seasonal distribution.—

Among the endemic places may be named Morocco, the Sahara (Biskra, Gafsa), Egypt, Crete, Cyprus, Asia Minor, Syria (Aleppo), Mesopotamia (Bagdad), Arabia, Persia, the Caucasus, Turkestan, India (Lahore, Multan, Delhi, etc.). Locally, Oriental sore is often called after some town or district in which it is specially prevalent; thus we have Delhi boil, Bagdad boil, and so forth. It is much more common in cities than in the country. In Bagdad few escape an attack; visitors, even of a few days only, are almost certain, at particular times of the year, to contract it. Juliano describes (*Journ. des Mal. Cut. et Syph.*, Oct., 1890) the disease as being common in Bahia, Brazil; the name Oriental sore, suggested by Tilbury Fox, is therefore no longer quite appropriate.

According to Hirsch, in the tropics this form of ulceration is specially prevalent about the commencement of the cool season, in more temperate climates towards the end of summer or beginning of autumn. Years of prevalence may be succeeded by years of comparative rarity; possibly in harmony with altered





sanitary conditions. In Delhi, for example, in 1864 from 40 to 70 per cent. of the resident Europeans were affected with the local sore; on certain sanitary improvements being effected, the frequency of the disease was immediately materially reduced.

Symptoms.—Oriental sore commences as a minute, itching papule which tends to expand somewhat as a shotty, congested infiltration of the derma. After a few days the surface of the papule becomes covered with fine, papery scales. At first these scales are dry and white; later they are moister, thicker, browner, and adherent. In this way a crust is formed which, on falling off, or on being scratched off, uncovers a shallow ulcer. The sore now slowly extends, discharging a scanty ichorous material which, from time to time, may become inspissated and crust over the sore which continues to spread underneath. The ulcer extends by the erosion of its perpendicular, sharp-cut, and jagged edge, which is surrounded by a slight or more considerable areola of congestion. The surface of the ulcer is irregular. Any granulations which form speedily break down. Subsidiary sores may arise around the parent ulcer, into which they ultimately merge. These sores, usually about an inch or so in diameter, in process of time may come, in some instances, to occupy an area several inches across.

After a variable period, ranging from two or three to twelve, or even more months, healing sets in. Granulation is slow and frequently interrupted. Often it commences at the centre whilst the ulcer may be still extending at the edge; often it is effected under a crust. Ultimately a depressed, white, or pinkish cicatrix is formed. Contraction of the scar, particularly if it happens to be on the face, may cause considerable and unsightly deformity.

Oriental sore may be single or multiple. Two or three are not uncommon; in rare instances as many as forty have been counted on the same patient.

They are mostly situated on uncovered parts—hands, feet, arms, legs, and, especially in young children, on the face; rarely on the trunk; never on the palms, soles, or hairy scalp.

In a very few instances the initial papule does not proceed to ulceration but persists as a scaling or scabbing, non-ulcerating, flattened plaque—just as sometimes happens in the case of the primary chancre of syphilis. Occasionally, from contamination with the virus of erysipelas, of sloughing phagedæna, or of some other infectious acute inflammatory skin disease, the primary lesion may become complicated and correspondingly modified and, perhaps, a source of serious danger. Otherwise, Oriental sore is troublesome and unsightly rather than painful or dangerous.

Histology; ætiology.—Section of the papule displays an infiltration of the derma by a mass of small round granulation cells. These lie between the elements of the tissues, particularly about blood-vessels, lymphatics, and sweat glands; towards the centre of the lesion they completely replace the normal structures. Various micro-organisms have been described in association with Oriental sore. By staining sections in gentian violet and afterwards partly decolourising in spirit, Cunningham and Firth found certain violet-stained bodies, varying in size and grouping, in a proportion of the infiltrating cells. These bodies Cunningham was inclined to regard as parasites. Riehl, however, looks upon them as the result of a hyaline degeneration of protoplasm, and advances a claim for certain micrococci which he found in great profusion in the granulation cells. Other observers have likewise found micrococci in the tissues; but, so far, inoculations from cultures have failed to produce the sores. As the disease has been proved to be communicable to the lower animals (as well as to man) by inoculation of the discharge from the surface of the ulcers, there can be little doubt that in Oriental sore the discharges contain the specific germ; and, as Riehl's micrococcus did not



1870 Nov. 1904.

Major General D. Lane, counselled me for
a while, & a month standing when he had
contracted for his dog. The dog had a scar
on its nose and on one of its forepaws. The right

communicate the true type of sore, it is probable that the germ has yet to be discovered.

In what way, under natural conditions, the parasite enters the tissues it is as yet impossible to state definitely. Not improbably it is conveyed by flies or other biting insects, and by them either inserted into the skin or applied to some pre-existing wound or sore.

As a rule, second attacks do not occur. Observing this, the Jews of Bagdad at one time practised on their young children Oriental sore inoculation, selecting for the purpose some part of the body habitually covered by the clothes. They designedly forestalled the infection by natural agencies, which experience had taught them to regard as inevitable; and thereby avoided an ugly scar on the faces, hands or arms of their children.

Neither race, nor sex, nor age, nor occupation, nor social condition influences susceptibility.

Treatment.—Some have advocated destruction of the primary papule, and even of the ulcer, by caustics or by the actual cautery. It is doubtful if such a measure would prove effective. A knowledge of the nature and natural progress of the disease suggests a protective and soothing, rather than an irritating line of treatment. A dressing with some mild antiseptic ointment, as of iodoform, boracic or salicylic acid, is indicated. Dr. Andrew Duncan informs me that in India he has seen these sores treated with great success by bandaging over them a piece of thin sheet lead. Tonics when the patient is anæmic or debilitated, attention to the general health, change of climate should the disease persist beyond the usual time for cicatrisation, are indicated.

SECTION V.—ANIMAL PARASITES AND ASSOCIATED DISEASES.

CHAPTER XXXIII.

FILARIASIS.

HE who would properly qualify himself for medical work in warm countries must acquire something more than a smattering of helminthology. The conditions of life in the tropics are such that not only are the careless natives extremely subject to verminous invasions of many descriptions, but the more carefully living and fastidious European is also constantly exposed to being victimised in the same way. Obscure diseases are very often the expression of some form of helminthiasis. Many diseases which formerly were mysterious and inexplicable can now with confidence be referred to "worms." The ability to recognise the presence of these parasites has, in some instances, enormously enhanced our powers of diagnosis, treatment, and prophylaxis. The physician, therefore, cannot afford any longer to ignore what, not many years ago, was regarded as but an unimportant department of natural history.

I. PARASITES OF THE CIRCULATORY AND LYMPHATIC SYSTEMS.

THE FILARIÆ SANGUINIS AND FILARIASIS.

History.—Our knowledge of this subject dates from the discovery by Demarquay, in 1863, of an embryo nematode—*Filaria nocturna*—in the milky fluid from a case of chylous dropsy of the tunica



vaginalis (p. 530). Later, in 1866, Wücherer found the same organism in the urine of a number of cases of chyluria. In 1870 Lewis made a similar observation in India. Two years later, in 1872, the latter observer discovered that the blood of man was the normal habitat of this embryo parasite which he named, accordingly, *Filaria sanguinis hominis*. Since that time the subject has rapidly expanded, and its great practical importance in tropical pathology is now slowly becoming recognised. Recently the writer has pointed out that Lewis's filaria is not the only blood-worm in man, and that the human circulation is the habitat of the embryos of no fewer than four, possibly of five or even more, distinct species of nematodes.

Nomenclature.—In consequence of the discovery of so many additional and similar blood parasites, it has been deemed advisable to modify the original name of Lewis's filaria. This I have proposed to call *Filaria nocturna*. The other filariæ of the blood I have named *Filaria diurna*, *Filaria perstans*, *Filaria demarquaii*, *Filaria ozzardi* (a doubtful species), and a sixth, which may or may not be connected with one of the two last, I have called *Filaria magalhæsi*, after its describer.

Their pathological importance.—Only two of these parasites, so far as we know at present, appear to have important pathological bearings—*Filaria nocturna*, the embryonic form of *Filaria bancrofti*, a mature parasite inhabiting the lymphatics of man; and *Filaria perstans*, the embryo of a parasite, the mature form of which has but recently been discovered. The pathological significance of the latter is still doubtful. There can be no question of the importance in tropical pathology of *Filaria nocturna* and of its parental form; there is abundant reason to believe that it is the cause of endemic chyluria, of various forms of lymphatic varix, of tropical elephantiasis arabum, and probably of other obscure tropical diseases.

FILARIA NOCTURNA (*Filaria bancrofti*).

Geographical distribution and prevalence.—The geographical distribution of *Filaria nocturna* is very extensive. It has been found as an indigenous parasite in almost every country throughout the tropical and subtropical world as far north as Spain in Europe, and Charleston in the United States of America; and as far south as Brisbane in Australia. In many places quite 10 per cent., and in other places half, of the population harbour it. I have ascertained that it is probable that one-third of the inhabitants of at least one district in India—Cochin—carry blood filariæ. I also find that in some of the South Sea islands, Samoa for example, fully one-half of the people are affected in this way. Thorpe has shown (*Brit. Med. Journ.*, Oct. 3rd, 1896) that in the Friendly Islands 32 per cent. of the people harbour the filaria. The table on p. 483, drawn up from a number of observations on the blood of the natives of certain tropical countries, will convey some idea of the wide area over which this and the other blood filariæ are distributed, and of the degree of their prevalence in the countries mentioned.

Demonstration of blood filariæ.—Whoever would investigate the subject of filariasis will find that, in order practically to comprehend the subject and provide himself with abundant material for observation, it is advisable to make a systematic examination of the blood of the inhabitants of some district in which elephantiasis is endemic. If this be done, the observer is sure to come across, sooner or later, cases in which *Filaria nocturna* abounds, and also of the diseases to which it gives rise.

Technique.—I commend the following procedure as likely to supply the investigator not only with material but also with much useful information. Let him visit late in the evening some hospital, or prison, or other establishment where he can have an opportunity of examining the inmates, and let him procure slides of the blood of, say, 100 persons. The slides are conveniently prepared by pricking the finger of each





person in turn, transferring large drops of the blood so obtained to ordinary microscope slips by simply dabbing the centre of the slip on the blood. The blood is then spread out with a needle so as to cover in a moderately thin film about a square inch of one surface of each slip. The slips are allowed to dry; they are then labelled and put aside. One preparation of this description may be made from each person, who should be selected simply as a representative of the general population and therefore irrespective of his being physically sound, or of his being the subject of any particular type of disease.

The slides may be examined in various ways, either at once or, if more convenient, weeks or months afterwards; if kept dry they do not spoil. A convenient plan is to dip the slides, without previous fixing, in a weak solution of fuchsine—about three or four drops of the saturated alcoholic solution to the ounce of water. They are left in the stain for about an hour, and then examined wet and without cover-glass. If the slides are old they may stain too deeply; in this case they may be partially decolourised in weak acetic acid—two or three drops to the ounce of water—and afterwards washed.

Another plan is to fix the blood-film with alcohol and then to stain by running on a few drops of saturated watery solution of methylene blue, washing off the superfluous stain after half a minute and, if necessary, decolourising with weak acetic acid; the wet slide is then examined with the microscope. Or, without previous fixing with alcohol, the slide, after it has dried, may be dipped for a few seconds in water so as to wash out the hæmoglobin, dried, and then, with or without fixing, stained with methylene blue, logwood, or other suitable stain.

An inch objective and a mechanical stage with a parallel movement will enable the investigator to pass rapidly in review the whole of the blood on the slide. In unfixed slides if filariæ are present they will be detected readily; the hæmoglobin of the blood corpuscles being dissolved out by the watery stain, the white blood corpuscles and any filariæ that may be present are the only coloured objects visible on the slide, and therefore at once catch the eye.

In any district in which the filaria is moderately common, out of 100 slides prepared in this way from as many persons, probably eight or ten will be found to contain the parasite. When filariæ have been detected, the persons from whom the parasite-bearing slides came may be used afterwards as a source of supply for further examinations and study.

Demonstration of living filariæ.—When it is desired to study the living filaria, all that is necessary is to make three or four ordinary wet preparations of the blood of a filaria-infected person—making them during the evening or night, and ringing the cover-glasses with vaseline so as to prevent the





slides from becoming dry. In such preparations the filariæ keep alive for a week, or even longer, and can readily be detected by their movements, an inch objective being used in the first instance as a searcher.

Permanent preparations.—Permanent preparations may be made by fixing very thin films of blood with alcohol or heat, staining with methylene blue, eosine, etc., and mounting in xylol balsam. It is generally advisable before fixing to wash out the hæmoglobin with water or very weak acetic acid. Logwood is perhaps the best stain; it brings out the sheath very distinctly, and picks out the nuclei. Double staining with eosine and logwood shows very well the structure of the musculo-cutaneous layer of the worm, in addition to other anatomical details.

Description of embryonic form.—Examined in fresh blood, *Filaria nocturna* (Fig. 51, *a*) is seen to be a minute, transparent, colourless, snake-like organism which, without materially changing its position on the slide, wriggles about in a state of great activity, constantly agitating and displacing the corpuscles in its neighbourhood. At first the movements are so active that the anatomical features of the filaria cannot be made out. In the course of a few hours the movement slows down, and then one can see that the little worm is shaped like a snake or an eel—that is to say, that it is a long, slender, cylindrical organism, having one extremity abruptly rounded off, the other for about one-fifth of the entire length gradually tapering to a fine point. On measurement, it is found to be about $\frac{1}{80}$ of an inch in length by $\frac{1}{3000}$ to $\frac{1}{3500}$ of an inch in diameter—about the diameter of a red blood corpuscle.

When examined with a low power, it appears to be structureless; with a high power a certain amount of structure can, on close scrutiny, be made out. In the first place, it can be seen that the entire animal is enclosed in an exceedingly delicate, limp, structureless sack, in which it moves backwards and forwards (Fig. 52, *a*). This sack, or “sheath,” as it is generally called, although closely applied to the body, is considerably longer than the worm it encloses; so that that part of the sack which for the time being is not

occupied is collapsed and trails after the head, or tail, or both, as the case may be. It can be seen also that about the posterior part of the middle third of the parasite, there is what appears to be an irregular



Fig. 51.—(a) *Filaria nocturna* $\times 300$; (b) *F. diurna*, Africa $\times 300$; (c) *F. demarquii*, St. Vincent $\times 300$; (d) *F. ozzardi* and *F. perstans*, British Guiana $\times 300$; (e) *F. perstans*, Africa $\times 300$.

aggregation of granular material which, by suitable staining, can be shown to be a viscus of some sort (b). This organ runs for some distance along the axis of the worm. Further, if a high power be used, a closely set, very delicate transverse striation can be detected in the musculo-cutaneous layer throughout the entire length of the animal. Besides this, if carefully looked



for at a point about one-fifth of the entire length of the organism backwards from the head end, a shining, triangular V-shaped patch (*c*) is always visible. What

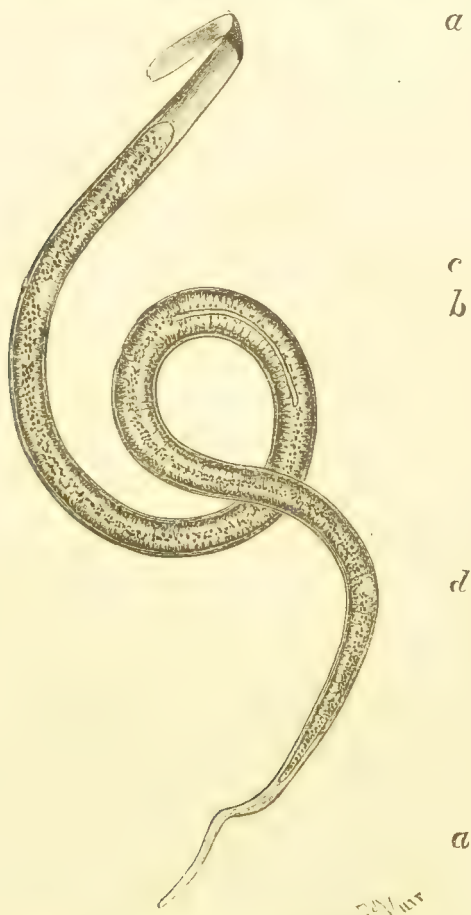


Fig. 52.—Anatomy of *Filaria nocturna*.

a a, Sheath ; *b*, central viscus ; *c*, V spot ; *d*, tail spot.

may be this V spot is brought out by very light staining with dilute logwood. The dye brings out yet another spot (*d*), similar to the preceding, though very much smaller ; this second spot is situated a short distance from the end of the tail. The former I have

designated the "V spot," the latter the "tail spot." These spots are probably connected with development, the V spot being the rudiment of the future water-vascular system or, perhaps, of the generative organs, the tail spot that of the anus, or cloaca and posterior part of the alimentary canal. The spots (*c*, *d*) are not stained by strong logwood or by the aniline dyes. Staining with logwood also shows that the body of the little animal is principally composed of a column of closely packed, exceedingly minute cells enclosed in the transversely striated musculo-cutaneous cylinder; at all events, many nuclei are thereby rendered visible. Dr. Low has recently pointed out to me that the break seen in all stained specimens in the central column of nuclei occurs at a point slightly posterior to the anterior V spot. This break can only be recognised in stained preparations.

When the movements of the living filaria have almost ceased, by careful focussing it can be seen that the head end is constantly being covered and uncovered by a six-lipped—or hooked—and very delicate prepuce; and, moreover, one can sometimes see a short fang of extreme tenuity suddenly shot out from the uncovered extreme cephalic end, and as suddenly retracted (Fig. 53, *c*, *d*).

Filarial periodicity.—A singular feature in the life of the embryo filaria is what is known as "filarial periodicity."



Fig. 53. Structure of head end of *Filaria perstans* (*a*, *b*) and of *F. nocturna* (*c*, *d*).





If under ordinary conditions of health and habit the blood be examined during the day, the parasite is rarely seen, or, if it be seen, only one or two specimens at most are encountered in a slide. It will be found, however, that as evening approaches, commencing about 5 or 6 o'clock, the filariæ begin to enter the peripheral circulation in gradually increasing numbers. The swarm goes on increasing until about midnight, at which time it is no unusual thing to find as many as three hundred, or even six hundred, in every drop of blood; so that, assuming that the parasites are evenly distributed throughout the circulation, it may be inferred that as many as forty or fifty millions are simultaneously circulating in the blood-vessels. After midnight the numbers begin gradually to decrease; by 8 or 9 o'clock in the morning the filariæ have disappeared from the peripheral blood for the day. This diurnal periodicity is, under normal conditions, maintained with the utmost regularity for years. Should, however, as Mackenzie has shown, a filarial subject be made to sleep during the day and remain awake at night, the periodicity is reversed; that is to say, the parasites come into the blood during the day and disappear from it during the night. It cannot be the sleeping state, as some have conjectured, that brings about this periodicity; for the ingress of the filariæ into the peripheral blood commences three or four hours before the usual time for sleep, and the egress several hours before sleep is concluded, and this egress is not complete until several hours after the usual time of waking.

No reasonable explanation of the cause of filarial periodicity has been given yet. A recent opportunity has enabled me to ascertain that, during their diurnal temporary absence from the cutaneous circulation, the filariæ retire principally to the larger arteries and to the lungs (Fig. 54), where, during the day, they may be found in enormous numbers.

The patient on whom this observation was made was the subject of lymph scrotum and varicose groin glands.

His blood contained large numbers of embryo filariæ, which exhibited the anatomical characters and periodicity characteristic of *F. nocturna*. One morning he committed suicide by swallowing a large quantity of dilute hydrocyanic acid. Death was almost instantaneous. At the *post-mortem* examination, made six hours later, a huge lymphatic varix was found occupying the pelvis and back part of the abdominal cavity. In the dilated lymphatic vessels many adult filariæ—*F. bancrofti*—were found. The distribution of the embryo filariæ was studied by counting them in drops of blood expressed from the various organs and also in sections. The result is summarised in the following tables:—

ENUMERATION OF FILARIÆ IN A DROP OF
BLOOD EXPRESSED FROM THE FOLLOWING
ORGANS.

Organ.	No. of Slides.	Aggregate No. of Filariæ.	Average per Slide.
Liver	3	2	$\frac{2}{3}$
Spleen	3	3	1
Brachial venæ comites .	4	111	28
Bone marrow	1	0	0
Muscle of heart . . .	3	365	122
Carotid artery . . .	1	612	612
Lung	10	6751	675

ENUMERATION OF FILARIÆ IN SECTIONS OF
THE FOLLOWING ORGANS.

Organs.	No. of Sections.	Aggregate No. of Filariæ.	Average per Section.
Liver	10	3	0.3
Spleen	4	0	0.0
Kidney	8	13	1.6
Brain	4	4	1.0
Muscle (voluntary) .	3	2	0.33
Heart muscle	4	68	17.0
Lung	6	301	50.16
Lobe of ear	4	1	0.25
Scrotum	4	0	0.0





In the lung sections (Fig. 54) the filariæ lay outstretched or variously coiled in the vessels, large and small. In the heart muscle they lay along the capillaries between the fibres; in the kidneys they seemed specially to affect the malpighian tufts; a very few were found in the capillaries of the brain; vast numbers were found in smears from the inner surface of the carotid arteries. The preparations afforded no explanation as to how the filariæ contrive to maintain their position in the blood

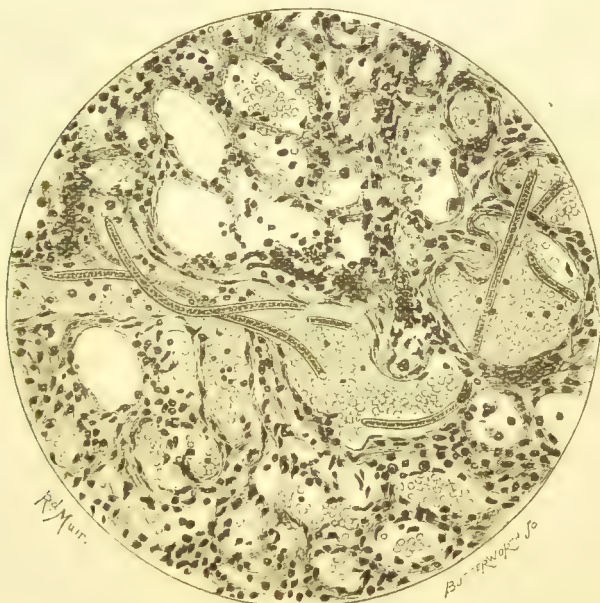


Fig. 54.—Section of lung, showing embryo filariæ in the blood-vessels.
(From a Microphotogram by Mr. Spitta.)

current, or as to the forces determining their peculiar distribution. Their absence from the liver and spleen is a remarkable fact.

The mosquito the intermediate host of *Filaria nocturna*.—Should the females of certain species of mosquito, probably belonging to the genus *Culex* [*C. pipiens* (?), *C. ciliaris*], which had fed on the blood of a filaria-infested individual be examined immediately after feeding, the blood contained in the stomach of the insects will be found to contain large numbers of living filariæ. If one of these mosquitoes

be examined three or four hours after it has similarly fed, it will be found that the blood corpuscles have, in great measure, parted with their hæmoglobin, and that the blood plasma in consequence of this and of dehydration has become thickened, though not coagulated. If attention be directed to the filariæ in

the thickened blood, it will be seen that many of them are actively engaged in endeavouring to escape from their sheaths. The diffused hæmoglobin has so thickened the blood plasma that it has become viscid, and holds, as it were, the sheath. This change in the viscosity of the blood seems to prompt the filariæ to endeavour to escape from their sheaths. They become restless and excited. Alternately retiring towards the tail end and then rushing forward to the head end of the sheath, the imprisoned parasite butts



Fig. 55.—Filarial ecdysis.

violently against the latter in frantic effort to escape. After a time, the majority succeed in effecting a breach and in wriggling themselves free from the sheaths which had hitherto enclosed them (Fig. 55).*

* This casting of its sheath by the filaria can be induced on ordinary blood slides by simply chilling them on ice, or by otherwise bringing about the diffusion of the hæmoglobin. The following method is usually successful: Ring with vaseline the cover-glasses of several ordinary wet preparations of finger blood obtained at night from a filarial patient; wrap the preparations





filaria now swims free in the blood, the character of its movements once more undergoing a remarkable change. Hitherto, though active enough in wriggling about, the parasite did not change materially its position on the slide; but now, having become free, it moves about from place to place—locomotes, in fact. If we dissect a mosquito at a somewhat later period after feeding, it will be found that the stomach of the insect, though still full of blood, contains very few



Fig. 56.—Section of thoracic muscles of mosquito, showing filariæ between the fibres: first day after the insect has fed on a filariated patient (From a Microphotogram by Mr. Spitta.)

filariæ, although their empty sheaths can be seen in abundance. If, however, we break up with needles the thorax of the insect and tease out in normal salt solution the muscular tissue, we shall find

separately in filter paper, and lay them, enclosed in a watertight tin box, on a block of ice, for six or eight hours—say overnight. Next morning examine them with the microscope. It will be found, as the chilled slides warm up, that wherever on the slides the hæmoglobin has become diffused and the blood lakey, the filariæ, as they gradually resuscitate from the chilling, begin to endeavour to break through their sheaths. By evening most of them have effected this, and their empty sheaths can be seen lying scattered about in the viscid blood. The blood must not be frozen.

what has become of the filariæ; we shall find that, after discarding their sheaths, they have quitted the stomach and entered the thoracic muscles of the insect, among which they can now be seen moving languidly (Figs. 56—59). By a course of serial dissections of filariated mosquitoes we can ascertain that in the thorax of the insect the parasite enters on a



Fig. 57.—Section of thoracic muscles of mosquito, seven days after it had fed on a filariated patient. (From a Microphotogram by Mr. Spitta.)

metamorphosis which takes from sixteen to twenty days (longer or shorter according to atmospheric temperature) to complete—a metamorphosis eventuating in the formation of a mouth, of an alimentary canal, and of a peculiar trilobed tail, as well as in an enormous increase in the size (to $\frac{1}{16}$ of an inch) and activity of the young parasite (Fig. 60, *a—f*). The filariæ now quit the thorax.

A few find their way to the abdomen, where, in properly prepared sections, they may occasionally be





seen in the tissues around the stomach, and even among the eggs at the posterior part of the abdomen. The vast majority pass forwards by the prothorax and neck and, entering the head, coil themselves up close to the base of the proboscis and beneath the pharynx and under-surface of the cephalic ganglia.



Fig. 58. Section of thoracic muscles of mosquito about twelve days after it had fed on a filariated patient. (From a Microphotogram by Mr. Spitta.)

Dr. Low, in sections prepared at the London School of Tropical Medicine, has shown recently that the filaria in its future progress finds its way into the proboscis, not by the salivary duct as is the case with the malaria parasite, nor along the buccal tube formed by the upper-surface of the hypopharynx and labrum, but by making a road for itself between the under-surface of the hypopharynx and upper-surface of the labium, where it can be readily recognised, lying free among the stilettes (Fig. 59 *a*). Apparently the filaria seeks to emerge (for such must be its object in entering

the proboscis) in pairs ; at all events, in those sections which I have examined, two worms were in each instance found together, their heads abreast of each other and close to the tip of the proboscis.

The parasites, it would seem, may remain in this position for an indefinite time, awaiting, it is to be



Fig. 59.—Section of mosquito about sixteen days after it had fed on a filariated patient. The filaria has escaped from the thoracic muscles. (From a Microphotogram by Mr. Spitta.)

presumed, an opportunity to enter a warm-blooded vertebrate host when the mosquito next feeds on such. Apparently the filariæ can discriminate between flesh and vegetable, for in mosquitoes fed on bananas they had not been deceived into passing into so inhospitable a medium, for up to forty days after the insect was infected, and after many meals of banana, they could still be found coiled up in the head or stretching along the proboscis.

These observations almost prove that, like the malaria parasite, the filaria is introduced into its





human definitive host by mosquito bite. Whether it may obtain an entrance by any other channel or medium it would be hard to prove and rash to deny. That the young filaria can live in water for a time is

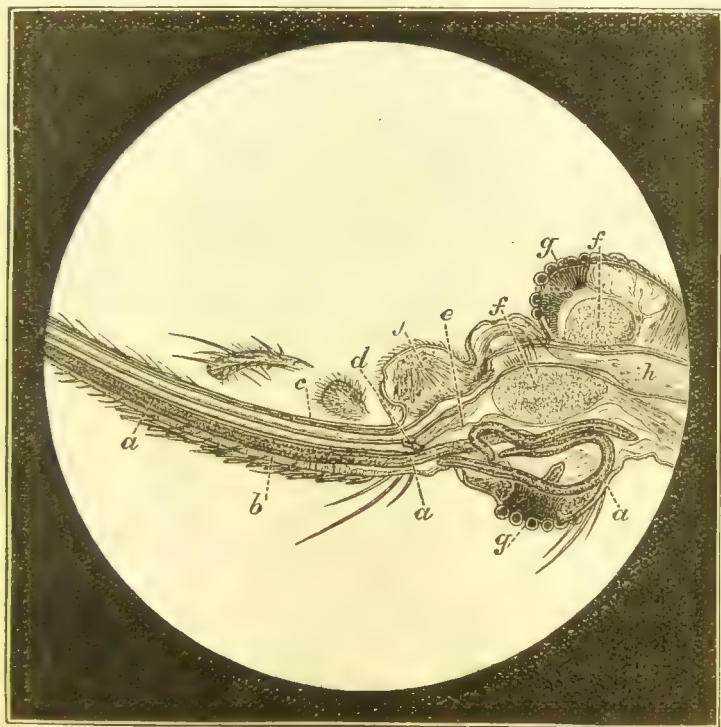


Fig. 59a.—*Filaria nocturna* in head or proboscis of mosquito.

a, a, a, Filaria; *b*, labium; *c*, labrum; *d*, base of hypopharynx; *e*, duct of veno-salivary gland; *f, f'*, cephalic ganglia; *g, g'*, eye; *h*, oesophagus; *j*, pharyngeal muscle.

certain; it is conceivable that some of them, such as those which at the completion of their stage of development in the mosquito find their way into the abdomen of the insect, may escape into water when the mosquito lays her eggs or dies.*

* In my original observations on this subject in 1879 and 1883 (Trans. Linnean Soc., 1883), I supposed that filarial metamorphosis, so far as the mosquito was concerned, is completed in from six to seven days. This, I now believe, was too short an estimate. Bancroft (*Journ. of Trop. Med.*, 1899) has lately shown

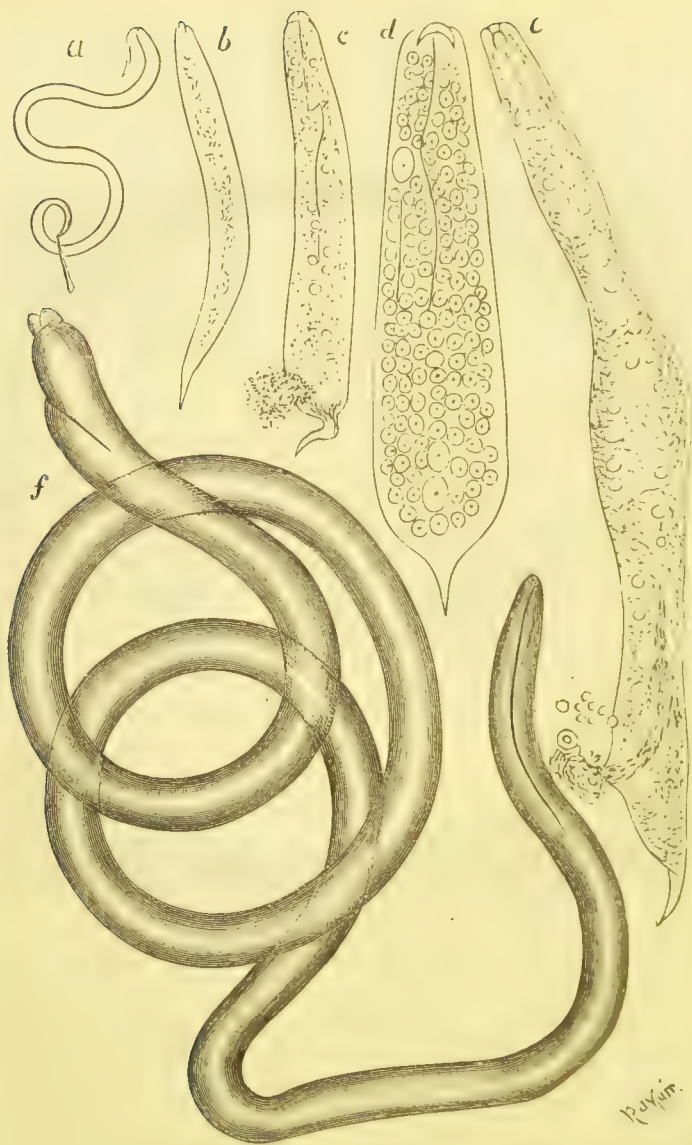


Fig. 60.—Metamorphosis of filaria in mosquito.

that it is necessary to feed the experimental mosquito (at all events, *C. ciliaris*), so as to keep it alive for at least sixteen days, to obtain the final stages of the metamorphosis. I have no doubt now that the insects on which my observations were made had fed





However introduced into the human body, the filaria finds its way into the lymphatics. Arrived in one of these, it attains sexual maturity, fecundation is effected, and in due course new generations of embryo filariæ are poured into the lymph. These, passing through the glands—if such should intervene, by way of the thoracic duct and left subclavian vein, or by the lymphatics of the upper part of the body—finally appear in the circulation.

Such is the life-history of *Filaria nocturna*; man is its definitive host, the mosquito its intermediary host. It is manifest that filarial periodicity is an adaptation of the habits of the parasite to the nocturnal habits of the mosquito. It is also manifest that the purpose of the “sheath” with which it is provided while cir-

repeatedly without my being aware of it, after their meal on the filariated patient. Bancroft has shown that by proper feeding (he uses banana for the purpose) mosquitoes may be kept alive for several months, any filariæ they happen to harbour remaining alive, but not advancing in development beyond the final stage alluded to in the above description. In the observations referred to I conjectured that the fully metamorphosed filaria escaped from the insect either at her death or when she deposited her eggs, and that thus, in drinking water, it obtained a chance of gaining access to the stomach of a human host. Before Low's observation Bancroft had very nearly guessed the truth, for he had expressed the idea that the filaria may be injected into man by the mosquito when it refeeds, or that it may be swallowed by man while it is still enclosed in its intermediary insect host.

Technique.—Mr. Max Colquhoun made for me beautiful sections illustrating the metamorphosis of the filaria in mosquito by the following technique. The insects, preserved in glycerine, were soaked in dilute acetic acid 5 per cent. for one day only; then in formalin 50 per cent., water 50 per cent. for one day; next in absolute alcohol for one day; *absolute* alcohol and ether, equal parts, one day; finally in celloidin for one day. They were then sectioned, placed in strong hematoxylin for two hours, decolourised in acid hydrochloric (1 per cent.) and alcohol, washed in water, passed through aniline oil, xylol, and mounted in xylol balsam. Paraffin sections are not a success.

In working with fresh mosquitoes, all that is necessary is to tease up the thorax of the insect with needles in normal salt solution, and, after removing the coarse fragments of the integuments of the mosquito, apply a cover-glass and place under an inch objective. The filariæ are readily detected. If the slide is dried, fixed and stained with a watery solution of some aniline dye, or with logwood, and mounted in xylol balsam, beautiful permanent preparations will easily be obtained.

culating in the human host is to muzzle the embryo filaria and prevent its breaking through the blood-vessels, and thereby missing its chance of gaining access to the mosquito. The cephalic armature is adapted for piercing the walls of the mosquito's stomach, and for enabling the parasite to travel through the tissues of the insect.

Parental forms (*Filaria bancrofti*). — The parent filariæ have been found many times. They are long, hair-like, transparent nematodes, three or four inches in length (Fig. 61). The sexes live together, often inextricably coiled about each other. Sometimes they are enclosed, coiled up several in a bunch, and tightly packed in little cyst-like dilatations of the distal lymphatics (Maitland); sometimes they



Fig. 61.—Parental form of *Filaria bancrofti*: female. (Natural size.)

lie more loosely in lymphatic varices; sometimes they inhabit the larger lymphatic trunks between the glands, the glands themselves, and, probably not infrequently, the thoracic duct.

The *female filaria* is the larger, both as regards length and thickness. The two uterine tubes, occupying the greater extent of her body, are filled with ova at various stages of development. In both sexes the oral end (Fig. 62, *b*) is slightly tapered, club-shaped, and simple; the tail (Fig. 62, *a*, *c*) also is tapered to comparatively small dimensions, its tip being rounded off abruptly. The vagina opens not very far from the mouth, the anus just in advance of the tip of the tail. The cuticle is smooth and without markings.

To the naked eye the *male worm* is characterised by its slender dimensions, by its marked disposition to curl, and by the peculiar vine-tendril-like tail, the extreme end of which is sharply incurvated (Fig. 62, *a*).* The cloaca gives exit to two slender, unequal spicules.

* According to Maitland this incurvation of the tip of the tail is a *post-mortem* phenomenon.





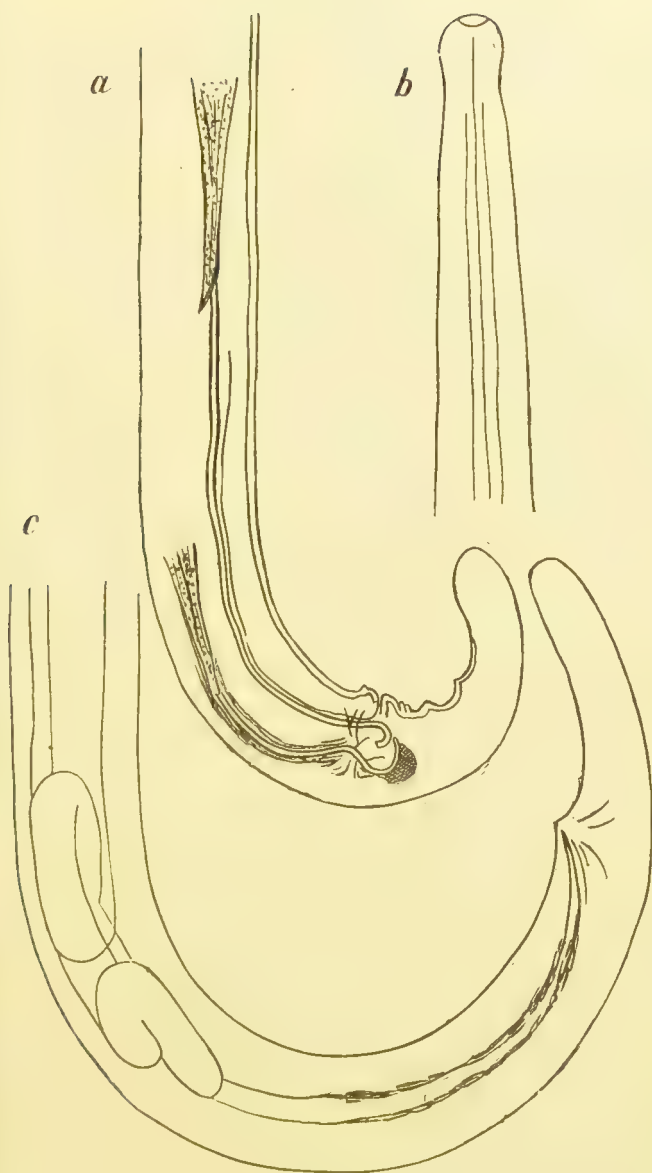


Fig. 62.—Parental forms of *Filaria bancrofti*.
 a Tail of male; b, head and neck; c, tail of female.

The existence of caudal papillæ in the male worm has not as yet been satisfactorily ascertained; analogy leads us to believe that such exist, but if they are present they must be exceedingly minute. (*See table, p. 535.*)

These parental forms were named by Cobbold *Filaria bancrofti*, after the late Dr. Bancroft, sen., of Brisbane, who was the first to find them.

Morbid anatomy and pathology.—*The filaria not generally pathogenetic.*—In most cases of filarial infection the parasite exercises no manifest injurious influence whatever. In a certain proportion of instances, however, there can be no doubt that it does have a very prejudicial effect on its host; and this mainly by obstructing lymphatics. The healthy, fully-formed embryo filariæ—that is to say, the filariæ which, by means of the microscope, we see in the blood—have, so far as we can tell, no pathological properties whatever; the parent worm and the immature products of conception, alone, are dangerous.

Filarial disease originates in injury of lymphatic trunks.—Roughly speaking, the filaria causes two types of disease; one characterised by varicosity of lymphatics, the other by more or less solid œdema. The exact way in which the parasite operates has not been definitely and absolutely ascertained for all types of filarial disease. Apparently, in some instances, a single worm, or a bunch of worms, may plug the thoracic duct and act as an embolus or originate a thrombus; or the worm may give rise to inflammatory thickening of the walls of this vessel, and so lead to obstruction from the consequent stenosis or thrombosis. In other instances the minor lymphatic trunks may be similarly occluded. As an effect of either form of occlusion, the lymphatic areas drained by the implicated vessels are cut off from the general circulation. There is then a rise of lymph pressure in the occluded vessels with consequent varicosity; or there may be lymphatic œdema; or there may be a combination of these.

Calvert (Bulletin of the Johns Hopkins
Hosp. June 1904) concludes that in the
early stages of filariasis there is leucocytosis
& eosinophilia which waxes during the
day time. This condition decreases as the
infection advances.





Pathology of lymphatic varix.—In consequence of the rich anastomosis existing between the contiguous lymphatic areas, on filarial obstruction

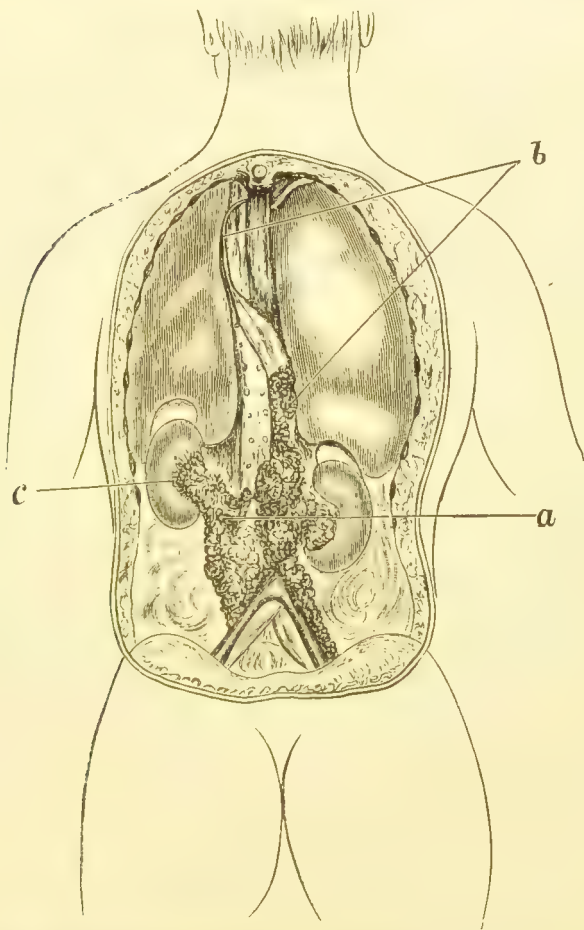


Fig. 63.—Dissection of the lymphatics in a case of chyluria. (*Mackenzie, Trans. Path. Soc., Lond.*)

a Dilated right renal lymphatics; *b*, thoracic duct; *c*, dilated left renal lymphatics

occurring in one of them a compensatory lymphatic circulation is sooner or later established. But before this can be properly effected a rise of lymph pressure and a dilatation of the lymphatics in the

implicated area must take place. This leads to lymphatic varix of different kinds, degrees, and situations. When the seat of filarial obstruction is the thoracic duct, the chyle poured into that vessel can reach the circulation only by a retrograde movement; consequently, this fluid may be forced to traverse in a retrograde way the abdominal and pelvic lymphatics, the lymphatics of the groin, scrotum, and abdominal wall. As a consequence, these vessels, together with the thoracic duct up to the seat of obstruction, become enormously dilated. In dissections of such cases (Fig. 63) the thoracic duct has been found distended to the size of a finger, the abdominal and pelvic lymphatics forming an enormous varix, perhaps a foot in diameter and many inches in thickness, concealing kidneys, bladder, and spermatic cords. In such cases, when one of the vessels of the varix is pricked or ruptures, the contents are found to be white or pinkish. They are not limpid like ordinary lymph. They are chyle therefore; chyle on its way to enter the circulation by a retrograde compensatory track. When the varix involves the integuments of the scrotum, the result is "lymph scrotum"; when most prominent in the groin, then a condition of glands is produced which I have called "varicose groin glands"; when the lymphatics of the bladder or kidneys are affected and rupture from over-distension, then chyluria is the result; when those of the tunica vaginalis rupture, then there is chylous dropsy of that sac—"chylocele"; the same may happen in the peritoneum—chylous ascites. Occasionally varicose lymphatic glands, resembling those frequently encountered in the groins, are found in the axilla. Occasionally limited portions of the lymphatic trunks of the limbs are similarly and temporarily or more permanently distended. This, doubtless, is the pathology of all those forms of filarial disease characterised by visible varicosity of lymphatics, with or without lymphorrhagia.

Filariae may disappear from the blood in such





cases.—In filarial disease associated with lymphatic varix embryo filariæ are generally present in the blood as well as in the contents of the dilated vessels. Sometimes, it is true, the embryo is not found. Such cases are probably of long standing; had the filaria been looked for at the commencement of the disease, it would have been discovered. I have watched cases in which the embryo has disappeared in this way; though at first found in abundance in the blood, after a year or longer it ceased to appear there. The reason for this disappearance is doubtless the death of the parent parasites, an occurrence I have seen associated with an attack of lymphangitis. It must be borne in mind that, although the original cause of the varix may disappear, the effects of its operation are permanent.

Pathology of elephantiasis arabum.—

Embryos not usually present in the blood in elephantiasis.—In those cases of filaria disease in which elephantiasis arabum is the leading feature, it is not usual at any stage of the established disease to find filariæ in the blood or elsewhere, unless it be in countries in which filariasis is extremely common and reinfection, or extensive infection, highly probable.

Reasons for regarding elephantiasis as a filarial disease.—From this circumstance—the absence of filaria embryos from the blood in elephantiasis—the question naturally arises, Why attribute this disease to the filaria? The answer to this is: (1) The geographical distribution of *Filaria nocturna* and of elephantiasis arabum correspond; where elephantiasis abounds there the filaria abounds, and *vice versa*. (2) Filarial lymphatic varix and elephantiasis occur in the same districts, and frequently concur in the same individual. (3) Lymph scrotum, an unquestionably filarial disease, often terminates in elephantiasis of the scrotum. (4) Elephantiasis of the leg sometimes supervenes on the surgical removal of a lymph scrotum. (5) Elephantiasis and lymphatic varix are essentially diseases of the lymphatics. (6) Filarial

lymphatic varix and true elephantiasis are both accompanied by the same type of recurring lymphangitis. (7) As filarial lymphatic varix is practically proved to be caused by the filaria, the inference that true elephantiasis—the disease with which the former is so often associated and has so many affinities—is attributable to the same cause, appears to be warranted.

Explanation of the absence of filariæ in the blood in elephantiasis.—If the filaria be the cause of tropical elephantiasis, how account for the absence of filaria embryos in the blood, as is the case in the vast majority of instances of this disease? The answer to this is:—Either the disease-producing filariæ have died; or the lymphatics draining the affected area are so effectually obstructed by the filaria, its products, or its effects, that any embryo filariæ they may contain, or may have contained, cannot pass along these vessels to the circulation.

We have already seen that in filarial lymphatic varix the parasite which produced the disease may die, particularly during attacks of lymphangitis; the same may occur in elephantiasis, and I believe that this does happen. I do not think, however, that this is the entire explanation.

Lymph stasis produced by filaria ova.—I have twice in filariasis found ova of the filaria in lymph; once in the lymph from a lymph scrotum, once in lymph procured by aspirating a varicose groin gland. Therefore, at times, the filaria may produce ova instead of swimming embryos. The ova of the filaria are not like the long, supple, slim, active, swimming embryo; they are passive, more or less rigid, oval bodies nearly five times the diameter of the embryo coiled up in their interior. In consequence of their size and passive character the ova, unlike the normal free swimming embryos, are quite incapable of traversing such lymphatic glands as, in the event of their escape from the parental worm, they may be passively carried to by the lymph stream. It is an





accepted fact in pathology that an essential element in the causation of elephantiasis is lymph stasis. I have ventured to conjecture that the stasis of lymph which eventuates in *tropical* elephantiasis is produced by embolism of the lymphatic glands by ova of the filaria.

But, it may be asked, why should ova enter the lymph stream? In normal conditions the embryo filaria at birth is already fully outstretched. If ova are expelled by the parent filaria, it must be as a result of some hurrying of the process of filarial parturition. That this does occur sometimes the discovery, already mentioned, of filaria ova in the lymph fully proves; and I believe that this is really what does occur, and that it is the initial step in the development of filarial elephantiasis. We can readily understand how, in consequence of mechanical injury, to which from her exposed position in the limbs she must be frequently subjected, or of some other cause, the parent filaria may miscarry. Should this happen, then the contents of her uterus will be expelled prematurely and before the ova normally lying at the upper part of her uterus have become the long, outstretched, active embryos we see in the blood. If a crowd of these passive, massive ova are carried by the lymph stream to the lymphatic glands of a limb, in the lymphatic trunks of which a female filaria is lying aborting, embolism of the afferent lymphatics of the glands must result and stasis of lymph in the limb ensue.

Inflammation necessary for the production of elephantiasis.—Lymph stasis alone does not produce elephantiasis; this has been proved by experimental ligature of lymphatic trunks. It may produce a form of œdema, but not true elephantoid hypertrophy. Should inflammation occur in an area of lymphatic congestion so produced, as it is very apt to do on the slightest injury, then elephantiasis will supervene; for, unless the lymphatics of an inflamed area are patent, the products of inflammation are not completely

absorbed. Erysipelatoid inflammation, frequently recurring, is a well-recognised feature in almost every case of elephantiasis arabum.

Sequence of events in elephantiasis.—The sequence of events in the production of elephantiasis is, I believe, as follows:—Parent female filaria in the lymphatic system of the affected part; injury of the filaria; premature expulsions of ova in consequence of injury; embolism of lymphatic glands by ova; stasis of lymph; lymphangitis from subsequent traumatism or other cause in the congested area; imperfect absorption of the products of inflammation; recurring attacks of inflammation leading to gradual, intermittingly progressive, inflammatory hypertrophy of the part.

In this way I explain the production of elephantiasis by the filaria. And in this way I explain the absence from the blood of the embryos of the parasite which started the disease; they cannot pass the occluded glands. Very likely the parent worm dies at an early stage of the disease; killed by the cause which led to premature parturition, or by the subsequent lymphangitis.

The subjects of elephantiasis less liable than others to filariæ in the blood.—Some years ago I made a curious observation which supports the view just stated. I received from Surgeon-Major Elcum eighty-eight slides of night blood from eighty-eight natives of Cochin. Of these eighty-eight persons fourteen were affected with elephantiasis, seventy-four were not so affected. Of the slides coming from the seventy-four non-elephantiasis cases twenty contained filariæ, about one in every three and a half; of the fourteen elephantiasis cases only one had filariæ. Why should the elephantiasis cases have proportionately fewer filariæ than the non-elephantiasis cases? Because in the former the existence of elephantiasis implied that a large area of their lymphatic systems was blocked, and that the blood could be stocked with embryo filariæ carried by the lymph from only

a relatively small lymphatic area; there was, therefore, a proportionately lesser likelihood of the presence of parent filariæ having for their young an unobstructed passage to the blood.

FILARIAL DISEASES.

Enumeration of the filarial diseases.—The diseases known to be produced by *Filaria nocturna* are abscess; lymphangitis; varicose groin glands; varicose axillary glands; lymph scrotum; cutaneous and deep lymphatic varix; orchitis; chyluria; elephantiasis of the leg, scrotum, vulva, arm, mamma, and elsewhere; chylous dropsy of the tunica vaginalis; chylous ascites; chylous diarrhœa, and probably other forms of disease depending on obstruction or varicosity of the lymphatics, or on death of the parent filaria.

Abscess.—Occasionally, as already mentioned, whether in consequence of blows or other injuries, of lymphangitis, or of unknown causes, the parent filaria dies. Generally the dead body is absorbed, just as a piece of aseptic catgut would be. Sometimes the dead worm acts as an irritant and causes abscess, in the contents of which fragments of the dead filaria may be found. Such abscesses, occurring in the limbs or scrotum, will discharge in due course, or may be opened; they lead to no further trouble. Should they form in the thorax or abdomen, serious consequences and even death may ensue.

Probably, in certain instances, abscess forms at times independently of the death of the parasite—in varicose glands, in lymph scrotum, in elephantiasis, or in similar areas of weakened tissues and lymphatic congestion.

The death of the parental filariæ is apt to be lost sight of as a possible cause of abscess in the subjects of filarial infection. Deep-seated pain in the thorax or abdomen, with inflammatory fever followed by hectic, and a diminution in the number of embryo filariæ in, or their entire disappearance from, the

peripheral blood should, in such circumstances, suggest a diagnosis of filarial abscess and indicate exploration and, if feasible, active surgical interference.

Lymphangitis and elephantoid fever.—

Symptoms.—Lymphangitis is a common occurrence in all forms of filarial disease, particularly in elephantiasis, varicose glands, and lymph scrotum. When occurring in the limbs the characteristic painful, cord-like swelling of the lymphatic trunks and associated glands, and the red congested streak in the superjacent skin, are usually apparent at the commencement of the attack. Very soon, however, the connective tissue and skin of the implicated area become inflamed and tense, and high fever, preceded by severe and prolonged rigor, sets in. The attack may continue for several days, and is accompanied by severe headache, anorexia, often vomiting, and sometimes delirium. After a time the tension of the inflamed integuments may relieve itself by a lymphous discharge from the surface. Usually the attack ends in profuse general diaphoresis. The swelling then subsides gradually though not entirely, the parts remaining permanently somewhat thickened. Lymphangitis may be confined to groin glands, testis, spermatic cord, or abdominal lymphatics.

Diagnosis.—This fever, appropriately named by Fayrer “elephantoid fever,” occurs habitually, at varying intervals of weeks, months, or years, in nearly all forms of elephantoid disease. Its tendency to recur, the severe rigor ushering it in, and the terminal diaphoresis cause it to be mistaken for ague. The implication of the lymphatics, the local pain, the erysipelatoid swelling, the prolonged pyrexial stage, the absence of the *Plasmodium malariae* from the blood, the presence there very possibly of the filaria, and the powerlessness of quinine to control the fever, suffice for diagnosis. Nevertheless, error in diagnosis is common, particularly when the attacks recur frequently, and with a certain degree of regularity, as is usually the case.

Treatment.—The treatment should consist in removing any cause of irritation, and in rest, elevation of the affected part, cooling lotions or warm fomentations, mild aperients, opium to relieve pain, and, if tension



Fig. 64.—Varicose groin glands and chylocele. (From a patient under the care of Mr. Johnson Smith.)

is great, pricking or scarifying the swollen area under suitable aseptic conditions.

Varicose groin glands (Fig. 64).—*Associated conditions.*—Varicose groin glands are frequently associated with lymph scrotum; sometimes with chylous dropsy of the tunica vaginalis; sometimes with chyluria. Occasionally all four conditions co-exist in the same individual.

Symptoms.—As a rule, commencing painlessly, the patient is not aware of the existence of these varicose

glands until they have attained considerable dimensions. Then, a sense of tension, or an attack of lymphangitis, calls attention to the state of the groins, where certain soft swellings are discovered. These swellings may be of insignificant dimensions, or they may attain the size of a fist. They may involve both groins, or only one groin; they may affect the inguinal glands alone, or the femoral glands alone, or, and generally, both sets together.

To the touch they feel soft, doughy, and obscurely lobulated. The skin, natural in appearance, can be glided over the surface, but the tumours themselves are not movable over the subjacent fascia. Occasionally hard, kernel-like pieces can be felt in their interior, or the entire mass may be more or less indurated. On thrusting a hypodermic needle into the swellings, white or reddish chylous, or clear lymphous fluid can be aspirated in abundance. This fluid coagulates rapidly and usually contains living filaria embryos.

Diagnosis.—It is important to be able to diagnose these tumours from hernia, for which they are often mistaken. This can be done by observing that they are not tympanitic on percussion; that though pressure causes them to diminish, they do so slowly; that there is no sudden dispersion accompanied by gurgling, as in hernia, on taxis being employed; that they convey a relatively slight, or no impulse on coughing; that they slowly subside on the patient lying down, and slowly return, even if pressure be applied over the saphenous or inguinal openings, on the erect posture being resumed. The cautious use of the hypodermic needle will confirm diagnosis; which would be further strengthened by the co-existence of lymph scrotum, chyluria, or chylous hydrocele, and the presence of filariæ in the blood. *Chronic swellings about the groin, cords, testes, and scrotum in patients from the tropics should always be regarded as being possibly filarial.*

Pathological anatomy.—On dissection these tumours are found to consist of bunches of varicose





lymphatics, and to form part of a vast lymphatic varix involving the pelvic and abdominal lymphatics.

Treatment.—Unless they give rise to an incapacitating amount of discomfort, and are the seat of frequent attacks of lymphangitis, varicose groin glands are best left alone. It must always be remembered that they are part of an anastomosis necessary to life. Should they be very troublesome and incapacitate for work, they may be removed (Maitland, *Brit. Med. Journ.*, 1897). In operating, strict aseptic methods must be practised, as septic lymphangitis readily occurs, and has frequently proved fatal in such cases. Excision is not always satisfactory, as it may be followed by lymphorrhagia at the seat of the wound, by excessive dilatation of some other part of the implicated lymphatic area, by chyluria, or by elephantiasis in one or both legs.

It is said (Azema) that these glands tend to diminish in size after forty. I cannot confirm this statement from personal observation.

Similar varicose dilation of the axillary glands is sometimes, though much more rarely, found. Bancroft designated these varicose axillary and groin glands “*helminthoma elastica*.”

Cutaneous and deeper lymphatic varices.

—Occasionally cutaneous lymphatic varices are met with on the surface of the abdomen, on the legs, arms, and probably elsewhere. Sometimes they are permanent; sometimes, when more deeply situated, they constitute little swellings which come and go in a few hours. I believe these latter often depend on the actual presence of parent filariæ in the tumour. Such varices are evidence of lymphatic obstruction. Filarial lymphangiectasis of the spermatic cord is not uncommon. The contents may be milky and chylous, or straw-coloured and lymphous, according to situation and connections.

Thickened lymphatic trunks. — Surgeon-Lieut.-Col. Maitland (*loc. cit.*) has frequently seen in Madras cases of lymphangitis in which, after the

initial swelling and inflammation had subsided, a line of thickening remained. On excising this thickened tissue and carefully dissecting it, he has found minute cyst-like dilatations of the lymphatic involved, and in these cysts adult filariæ coiled up, sometimes dead, sometimes alive. The lymphangitis, he believes, is caused in these cases by the death of the filariæ. Daniels has made similar observations in British Guiana. Such a case I recently saw under the care of Dr. Abercrombie at Charing Cross Hospital in London. On the subsidence of a filarial lymphangitis of the arm a thickening, about the size of a finger-tip, remained on the forearm. Believing that it contained adult filariæ, this thickening was excised by Mr. Young and placed in normal salt solution. Eight hours later the mass was carefully dissected, and a living female filaria, about four inches in length, was turned out. The parasite continued to live and swim about actively in the salt solution for nearly two hours (*Brit. Med. Journ.*, April 24th, 1897).

Lymph scrotum (Fig. 65).—*Symptoms*.—In this disease the scrotum is more or less enlarged. Though usually silky to the touch, on inspection the skin presents a few, or a large number of, smaller or larger lymphatic varices which, when pricked or when they open spontaneously, discharge large quantities of milky, or sanguineous-looking, or straw-coloured, rapidly coagulating lymph or chyle. In some cases eight or ten ounces of this substance will escape from a puncture in the course of an hour or two; it may go on running for many hours on end, soiling the clothes of the patient and exhausting him. Usually embryo filariæ can be discovered in the lymph so obtained, as well as in the blood of the patient. In a large proportion of cases of lymph scrotum the inguinal and femoral glands, either on one or on both sides, are varicose.

Probably provoked by injury in rubbing against the thighs and clothes, erysipelatoid inflammation

and elephantoid fever are frequent occurrences. Abscess is not uncommon. In time, in a proportion of cases, the scrotum tends to become permanently



Fig. 65.—Lymph scrotum and varicose groin glands. (From a photograph by Dr. Rennie, Foochow.)

thickened and to pass into a state of true elephantiasis.

Treatment.—Unless inflammation be a frequent occurrence, or there be frequent and debilitating lymphorrhagia, or unless the disease is tending to pass into true elephantiasis, lymph scrotum—kept

scrupulously clean, powdered, suspended, and protected—had better be left alone. Should, however, for these or other reasons, it be deemed expedient to remove the diseased tissues, this can be effected easily. The scrotum should be well dragged down by an assistant whilst the testes are pushed up out of the way of injury. A finger knife is then passed through the scrotum, and in sound tissues, just clear of the testes, and the mass excised by cutting backwards and forwards. No diseased tissues, and hardly any flap, should be left. Sufficient covering for the testes can be got by dragging on the skin of the thighs, which readily yields and affords ample covering. It is a very common, but a very great mistake to remove too little. As a rule, the wound, if carefully stitched and dressed antiseptically, heals rapidly.

In consequence of this sudden and violent interference with an extensive varix, of which that in the scrotum is but a part, chyluria and sometimes elephantiasis of a leg may supervene. The patient should be warned of this possibility.

Chyluria.—*Pathology.*—When a lymphatic varix in the walls of the bladder, the consequence of filarial obstruction in the thoracic duct, ruptures, there is an escape of the contents of the varicose lymphatics into the urine. Chyluria is the result.

Symptoms.—This disease frequently appears without warning; usually, however, pain in the back and aching sensations about the pelvis and groins—probably caused by great distension of the pre-existing lymphatic varix—precede it. Retention of urine, from the presence of chylous coagula, is sometimes the first indication of serious trouble. Whether preceded by aching, or by retention, or by other symptoms, the patient becomes suddenly aware that he is passing milky urine. Sometimes instead of being white the urine is pinkish or even red; sometimes white in the morning, it is reddish in the evening, or *vice versa*. Sometimes whilst chylous at

one part of the day, it is perfectly limpid at another. Great variety in this respect exists in different cases, and even in the same case from time to time, depending on temporary closure of the rupture in the lymphatic, and also on the nature of the food.*

Physical characters of chylous urine.—If chylous urine be passed into a urine glass and allowed to stand, as a rule, within a very short time, the whole of the urine becomes coagulated. Gradually the coagulum contracts until, at the end of some hours, a small, more or less globular clot, usually bright red or pinkish in colour, is floating about in a milky fluid. Later, the milky fluid separates into three layers. On the top there is formed a cream-like pellicle; at the bottom a scanty reddish sediment, sometimes including minute red clots; in the centre the mass of the urine forms a thick, intermediate stratum, milky white or reddish white in colour, in which floats the contracted coagulum. If a little of the sediment be taken up with a pipette and examined with the microscope, it is found to contain cells like red blood cells, lymphocytes, granular fatty matter, epithelium and urinary salts, and, mixed with these in a large proportion of cases though not in all, filaria embryos. The middle layer contains much granular fatty matter; whilst the upper cream-like layer consists of the same fatty material in greater abundance, the granules tending to aggregate into larger oil globules. If a small portion of the coagulum be teased out, pressed between two slides, and examined with the microscope, filariæ, more or less active, may be found in the meshes of the fibrine. If ether be shaken up with the milky urine, the fat particles are dissolved out and the urine becomes clear; the fat may be recovered by decanting and evaporating the

* The sanguineous appearance so frequently seen in chylous urine and in other forms of filarial lymphorrhagia, depends on the formation of blood corpuscles in lymph long retained in the varicose vessels, and is a result of the normal evolution of the formed elements in that fluid. There is no rupture of blood-vessels, as some have asserted.

ether which floats on the urine. Boiling the urine throws down a considerable precipitate of albumin.

Recovery and relapse.—Chyluria comes and goes in a very capricious manner. Sometimes the urine remains steadily chylous for weeks and months, and then suddenly, without obvious cause, becomes limpid and natural-looking, and free from fat or albumin. Later a relapse will occur, again to disappear after an uncertain time; and so on during a long course of years.

Retention of urine.—Retention of urine is not an unusual occurrence; it is produced by the formation of coagulum in the bladder. The retention usually gives way after a few hours of distress, worm-like clots being passed.

Constitutional effects.—Although chyluria is not directly dangerous to life, yet, being prolonged, it gives rise to pronounced anæmia, depression of spirits, and feelings of weakness and debility, and tends to incapacitate the patient for active, vigorous life.

Exciting causes of chyluria.—Chyluria is very liable to occur, either for the first time, or as a relapse, in pregnancy or after childbirth; the disturbance of the pelvic lymphatics in pregnancy, and the muscular efforts attending labour apparently causing rupture of pelvic lymphatics previously rendered varicose by filarial obstruction of the thoracic duct. In men, running, leaping, and violent efforts generally, are sometimes assigned as its cause; usually the exciting cause is not discoverable.

Treatment.—The treatment of chyluria should be conducted on the same lines as the treatment for inaccessible varix elsewhere; that is to say, by resting and elevating the affected part, and thereby diminishing as far as possible the hydrostatic pressure in the distended vessels. Many forms of medicinal treatment have been advocated. Because during treatment with some drug a chyluria has subsided, curative properties are apt to be attributed to the drug which was being taken at the time. The best results are got by sending the patient to bed, elevating the pelvis,

Struma of Scrofula lymphatic
Mackintosh describes a case in which
what appeared to be a scrofula
had got into the lymphatic glands on
both sides. They could be removed. The
resulting wound in one required 4 lines
of treatment.

restricting the amount of food and fluid—especially fatty food, gentle purgation, and absolute rest. It will be found that a day or two of treatment on these lines is often followed by temporary, perhaps prolonged, cessation of the chyluria. The drugs which have been particularly lauded in the treatment of this disease are gallic acid in large doses, benzoic acid in large doses, glycerine, the tincture of the perchloride of iron, decoction of mangrove bark, chromic acid, quinine, salicylate of soda, ichthyol, and *Nigella sativa*. I do not believe that these substances have any influence whatever in stopping the lymphorrhagia. Neither do I believe that thymol, recommended by Lawrie, or methylene blue, recommended by American writers, has any effect whatever, either on the filaria or on the disease it gives rise to; since their first recommendation both drugs have been tried, but in other hands have failed.

Filarial orchitis.—Several French writers describe under the name “malarial orchitis” a special form of inflammation of the testes, and here and there in Indian medical literature allusion is made to the same, or a similar subject. I have many times seen filarial orchitis, but I cannot say I have seen orchitis of purely and unquestionably malarial origin. The fever attending filarial orchitis—which is usually associated with lymphangitis of the spermatic cord and perhaps with inflammation of the scrotum, like ordinary elephantoid fever resembles very closely a malarial attack and may be mistaken for this. Without absolutely denying the existence of such a disease as malarial orchitis, I would suggest that the affection described by the French and Indian writers referred to, and endemic inflammations of testes, spermatic cords, and scrotum generally, are of filarial origin.

Filarial synovitis.—Maitland (*Ind. Med. Gaz.*, Oct., 1898) mentions acute synovitis of the knee-joint as one of the filarial diseases, and gives the particulars of five cases. He considers the concurrence

of synovitis with filarial invasion too common to be accidental.

ELEPHANTIASIS.

Its prevalence.—Elephantiasis is by far the most frequent manifestation of filarial infection, and is exceedingly common in some of the endemic districts. Thus in certain districts in Cochin about 5 per cent. of the population; in Samoa about every second individual; in Huahine seven-tenths of the adult male population are affected. In many other tropical and sub-tropical countries, if not so common as in those just mentioned, elephantiasis is, nevertheless, common enough.

Parts affected.—In 95 per cent. of the cases the lower extremities—either one or both—alone, or in combination with the scrotum or arms, are the seat of the disease. The foot and ankle only; or the foot and leg; or the foot, leg, and thigh; may, each or all, be involved. The scrotum is also a common situation for elephantiasis. The arms are more rarely attacked; still more rarely the mammæ, vulva, and circumscribed portions of the integuments of the limbs, trunk, or neck.

The recurring erysipelatoid attacks.—The disease in any of these situations commences with a rapidly evolved lymphangitis, dermatitis, and cellulitis, accompanied by elaphantoid fever. On the subsidence of the acute symptoms the skin and subcutaneous fascia of the affected part do not quite resume their original proportions; the inflammatory effusion not being completely absorbed, some permanent thickening remains. Recurrences of this inflammation once or twice a month, or perhaps once in six months, or every twelve months, or even at longer intervals, add a little each time to the bulk of the limb or scrotum: thus, gradually, an enormous swelling may be built up. Occasionally, though very rarely, enlargement may progress after one, two or more initial inflammatory attacks and without further recurrence of these.

Clinical characters of the swelling.—The affected part is greatly increased in bulk. The surface of the skin, in confirmed elephantiasis especially, is rough and coarse; the mouths of the follicles are sometimes unusually distinct; the papillæ and glands are either hypertrophied or atrophied; the hair is coarse and



Fig. 66.—Elephantiasis of legs; scrotum and right arm slightly affected. (From a photograph by Dr. Turner, Samoa.)

sparse; the nails are rough, thick, and deformed. Around joints the thickened integuments are thrown into folds, the comparatively smooth-sided and deep interlying sulci permitting limited movement. There is no distinct line of demarcation between healthy and diseased skin. The implicated integuments are hard, dense, pit but slightly if at all on pressure, and cannot be pinched up or freely glided over the deeper parts.

Its macroscopic anatomy.—On cutting into the swelling, the derma is found to be dense, fibrous, and enormously hypertrophied. The subjacent connective tissue is increased in bulk, having, especially in the case of the scrotum, a yellowish, blubbery appearance from lymphous infiltration. A large quantity of fluid wells out on division of such tissues. The muscles, nerves, and bones are not necessarily diseased, although in rare instances they may be degenerated and slightly or considerably atrophied from pressure. The blood-vessels are large; the lymphatics dilated; the associated lymphatic glands, both of the same side and very often of the opposite side, enlarged and dense.

True elephantiasis is permanent.—Though in recent cases elephantiasis of the limbs may be much reduced by treatment, the disease is never permanently recovered from. Lymphatic œdema in areas which have never inflamed subsides readily enough on pressure or elevation.

Elephantiasis of the legs (Fig. 66).—Elephantiasis of the lower extremities is usually, though by no means always, confined to below the knee. The swelling may attain enormous dimensions and involve the entire extremity, the leg or legs attaining a circumference, in aggravated cases, of several feet.

Treatment.—In the treatment of elephantiasis of the leg the patient should be encouraged to persevere with elastic bandaging, massage, and elevation of the limb. Ligature of the femoral artery has been practised; it is probably useless, and is certainly not a justifiable method of treatment. Sometimes, in extreme cases, good results are got from excision of redundant masses of skin, a longitudinal strip of three or four inches in breadth by a foot or more in length being dissected off. Electrolysis and mercury have also been used; I question their value. During the acute attacks, tension may be relieved by aseptic punctures with a sharp lancet. At all times the limb must be carefully guarded from injury, and shoes









and trousers worn. Slight injuries provoke the inflammatory recurrences. Wading in water, prolonged standing, violent exercise, and exposure to a hot sun are injurious, and should be avoided as far as possible.

Elephantiasis of the scrotum (Fig. 67).
Weight of tumours.—Elephantiasis of the scrotum, or



Fig. 67.—Elephantiasis of the scrotum : left leg slightly affected.
(From a photograph by Dr. Turner, Samoa.)

“scrotal tumour” as it is sometimes called, may attain an enormous size. Ten, fifteen, or twenty pounds are common weights for these tumours, and forty or fifty pounds is by no means uncommon ; the largest recorded weighed 224 lbs.

Anatomical characters.—There are certain points in the anatomy of scrotal tumour which the operating surgeon must bear in mind. These tumours consist of two portions (Fig. 68): First, a dense rind of hypertrophied skin (A *e*), thickest towards the lower part

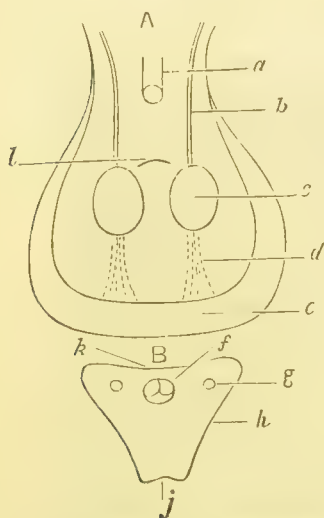


Fig. 68.—Diagram of the anatomy of elephantiasis of the scrotum.

and gradually thinning out as it merges above into the sound skin of the pubes and thighs; second, enclosed in this rind, a mass of lax, blubbery, dropsical, easily torn through, areolar tissue, in which testes, cords, and penis are embedded. The shape of the tumour is more or less pyriform. The upper part, or neck, on transverse section (B) is triangular, the base (B *k*) of the triangle being in front, the apex (B *j*)—usually somewhat bifid from dragging on the gluteal folds—towards the

anus, the sides (B *h*) towards the thighs. In the latter situation the skin, though usually more or less diseased, is, from pressure, softer and thinner than elsewhere, tempting the surgeon to utilise it for the formation of flaps—not always a wise proceeding. The penis (A *a*, B *f*) always lies in the upper and fore part of the neck of the mass; it is firmly attached to the pubes by the suspensory ligament. The sheath of the penis is sometimes specially hypertrophied, standing out as a sort of twisted ram's horn-like projection on the anterior surface of the tumour; this, however, is unusual. Generally, the sheath of the penis is incorporated in the scrotal mass, the prepuce being dragged on and inverted so as to form a long channel leading to the glans penis and opening (A *l*) half-way down, or even lower, on the face of the



tumour. The testes (A c), buried in the central blubbery tissue, usually lie towards the back of the tumour, one on each side, in large tumours generally nearer the lower than the upper part. They are more or less firmly attached to the under part of the scrotum by the hypertrophied remains of the gubernaculum testis (A d); a feature to be specially borne in mind by the surgeon. As a rule, both testes carry large hydroceles with thickened tunicae vaginales. The spermatic cords, also (A b, B g), are thickened and greatly elongated. The arteries supplying these enormous growths are of considerable size; the veins, too, are very large and, as they permit regurgitation of blood from the trunk, are apt to bleed freely.

Their importance.—Beyond inconvenience from their weight, the presence of the cumbersome mass between the legs, the suffering attendant on recurring attacks of inflammation and elephantoid fever, the sexual disability, and the unsightliness, these tumours are not of great importance; they do not, as a rule, directly endanger life. They may grow rapidly or slowly; they may attain a large size in two or three years, or they may be in existence for years and at the end of this time amount to little more than a slight thickening of the scrotum. Occasionally, in large tumours, portions of the mass become gangrenous, or abscess may form, and in this way life may be endangered; this is not usual, however.

Treatment.—Scrotal tumour, as soon as it becomes unsightly or inconvenient, should be removed. Often after thorough removal of all the diseased integuments, elephantoid fever, which before may have been frequent, ceases to recur.

Treatment preliminary to operation.—If the tumour is of considerable size, the patient should keep his bed for a day or two before operation, the mass being suspended so as to drain it of fluid and blood. It is thus rendered lax, and the operator is enabled to ascertain by palpation the position of the testes and, if such chance to be present, of hernia—

a not very unusual complication. The possibility of undescended testes should not be overlooked.

Operation.—Before making provision for the prevention of hæmorrhage the operator should mark out by shallow cuts the line at which he proposes to separate the tumour, care being taken that these guiding incisions run through and include only absolutely sound skin. If the latter precaution be neglected, disease is very liable to recur in the scar or flaps. First, the tumour is turned up and a shallow

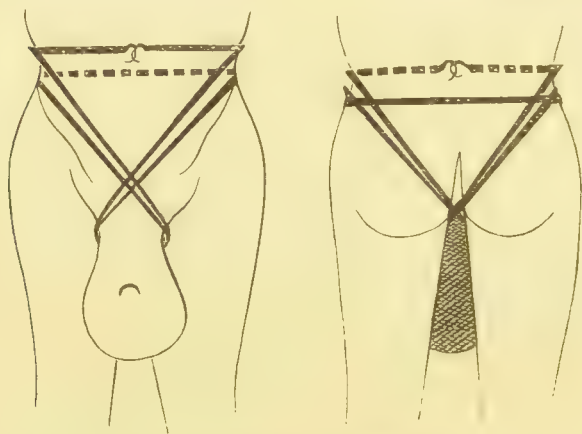


Fig. 69.—Rubber cord in position.

transverse cut is drawn in sound skin across the perineum in front of the anus. The tumour being allowed to fall down, a similar shallow cut is made across the pubes. The corresponding extremities of these two cuts are then united either by a straight cut, or, if there be a little sound skin on the thigh aspects of the tumour, by semilunar incisions.

Assistants then firmly draw down the scrotum as far as possible, and the surgeon, if he deem it desirable, applies elastic webbing over the mass so as to expel the blood it contains. Next, a stout rubber cord is wound, figure of 8 fashion, round the neck of the tumour, well above the guiding incisions, and over the pelvis, and firmly secured (McLeod) (Fig. 69). Or





the rubber cord is wound round the neck of the tumour only, being kept in place by four strips of bandage passing under the cord, one on each side of the scrotum before and behind, and firmly tied over another strip of bandage encircling the waist.

The testes and cord are first dissected out through long perpendicular incisions made in front, the remains of the gubernacula testis being hooked up with the finger and snipped through with scissors. The channel of the prepuce is next slit up, the incision being carried up to the pubic limiting mark. The penis can then be shelled out, the prepuce being first cut through around the corona glandis. If lateral flaps can be formed of sound skin they are then dissected up. The perineal and pubic incisions are now deepened and, assistants holding the testes and penis well out of the way, the neck of the tumour is cut through close to the perineum and pubes. Gaping vessels are all carefully ligatured, and redundant tunica vaginalis—if hydroceles be present—excised. The rubber cord is then removed.

When hæmorrhage has been controlled, the posterior halves of the flaps are brought together by sutures, the anterior halves being united over the testes to the pubic cut. The penis will therefore emerge from the point where the horizontal line meets the perpendicular line of what is now a T- or Y-shaped wound. If no flaps have been made, the testes may be fixed by stitching any tag of tissue connected with them to the perineum, and the dimensions of the wound reduced as much as possible by stitching up the corners at the pubes and perineum.

In dressing it is of importance that the raw surfaces be covered by some aseptic *non-fibrous* protective—such as oiled silk—before the antiseptic dressing is applied. A fibrous dressing next the raw surfaces is troublesome, as it sticks to the wound and is painful and difficult to remove. The dressing should be massive, well padded, and kept in place by an eight-tailed bandage secured in front and behind to a strap round

the waist, a hole being cut in front for the penis to emerge. The large wound generally does well. Skin grafting should be practised freely and early, especially round the root of the penis.

Mortality from operation.—The mortality from these formidable-looking operations, if they are carefully done, is small, and need not exceed 5 per cent. The results are very satisfactory, as a rule, the functions of the organs being retained or restored.

Elephantiasis of the arms.—This is comparatively rare. Allowing for the differences between the upper and lower extremities as regards gravitation of fluids, the symptoms and pathology of elephantiasis of the arm are the same as those of elephantiasis of the legs. Beyond the judicious employment of massage and elastic bandaging, little can be done in the way of treatment.

Elephantiasis of the vulva and mammæ.—Elephantiasis of the vulva (Fig. 70) and mammæ (Fig. 72) is still rarer. Where growth has become inconveniently large the diseased tissues should be removed. Instances are on record in which the integuments of the mammæ have become so thickened, heavy and elongated, that the organ has descended to the pubes and even to the knee. One such tumour weighed twenty-one pounds after removal. Tumours of the labia or of the clitoris, similarly, may attain a great size—eight or ten pounds, or even more.

Elephantiasis of limited skin areas.—Dr. Corney, of Fiji (*Lancet*, April 6th, 1889), states that pedunculated elephantoid tumours, springing from the groin or from the anterior surface of the thigh, are not uncommon in Fiji. One such tumour which he removed weighed twenty pounds. Dr. Daniels (*Brit. Guiana Med. Ann.*, 1896) has seen, both in Fiji and in Demerara, several cases of this description. (Fig. 71.) Dr. Silcock (*Indian Med. Gaz.*, July, 1895) describes a pedunculated tumour of this nature which he removed from the neck of an East Indian, and which weighed, after removal, thirty pounds. I have









FIG. 70.—ELEPHANTIASIS OF VULVA.

(From a photograph by Dr. Walter H. B. Macdonald.)

often seen limited areas of elephantoid thickening of the skin, particularly on the thighs. These conditions are easily dealt with by simple operations.

Chylous dropsy of the tunica vaginalis, and of the peritoneum; chylous diarrhœa.

Chylous dropsy of the tunica vaginalis is not an



FIG. 71.—PEDUNCULATED GROIN ELEPHANTIASIS.

(From a photograph by Dr. Daniels, Demerara.)

unusual occurrence in the tropics. A fluctuating swelling of the tunica vaginalis, which does not transmit light, and which is associated possibly with lymph scrotum, with varicose groin glands, with chyluria, or with filariæ in the blood, would suggest a diagnosis of this condition. These collections of chylous fluid in the tunica vaginalis generally contain





FIG. 72.—ELEPHANTIASIS OF MAMMÆ: LEFT LEG AND FOOT ALSO AFFECTED.

(From a photograph by Dr. Davies, Samoa.)

enormous numbers of filaria embryos. They may be treated as ordinary hydroceles, either by aseptic incision or by injection.

Filarial orchitis with effusion into the tunica vaginalis, according to Maitland is best treated by incision of the tunica vaginalis, turning out any clot that may be found in the sac, and stuffing the latter with iodoform gauze. This procedure, he says, gives immediate relief.

Chylous dropsy of the peritoneum, and chylous diarrhœa of filarial origin are very rare.

Prophylaxis of filarial disease.—The prevention of filarial disease resolves itself into protection from mosquito bites and securing a water supply uncontaminated by these insects. With this in view, unprotected wells, tanks, or stagnant pools must not be permitted in the neighbourhood of dwelling-houses or used as sources of drinking water; tanks and buckets for storing domestic water (the influence of these in spreading filarial disease has been ably demonstrated by Daniels in Demerara, *loc. cit.*) should be covered with fine wire netting, or regularly emptied and cleaned every three or four days. Water that is not absolutely beyond suspicion should be boiled or filtered.

The subjects of filariasis should be regarded as dangers to themselves and to the community, and be compelled to sleep under mosquito nets.

FILARIA DIURNA. (Fig. 51, b.)

I have twice encountered in negroes a blood worm with the same dimensions and anatomical characters, so far as these have been made out, as *F. nocturna*, but differing from this latter parasite inasmuch as it comes into the blood during the day and disappears from it during the night. One of these patients came from Old Calabar, the other from the Congo. The periodicity observed by the parasite was thoroughly made out by prolonged observation in one of the cases. As the man was in good health at the



time, and was observing ordinary habits as regards the hours of sleeping and waking, there can be little doubt that the parasite was not *F. nocturna*. Some years previously this patient had a *F. loa* in one of his eyes; it is just possible, therefore, that *F. diurna*, as I name this blood worm, is the embryonic form of the sexually mature *F. loa* (p. 559). This is merely a conjecture. I have no further observations to support it; indeed, the negative results as far as finding embryo filariæ in the blood in four cases of *F. loa* which I have examined, are against it.

Nothing is known about its life-history, or pathological significance. From recent observations I believe it to be very common (1 in 4) in certain districts on the lower Niger, where it seems to take the place among the natives that *F. perstans* holds among the Congo negroes.

FILARIA DEMARQUAII. (Fig. 51, c.)

In examining blood sent me by Dr. Newsam from natives of St. Vincent, West Indies, I found this blood worm in several individuals—in 10 out of 152 examined. It resembles *F. nocturna* and *F. diurna* so far as shape is concerned, but differs from them in size. I have had no opportunity of making trustworthy measurements of living specimens in suitably prepared slides, but, judging from rough preparations, *F. demarquaii* appears to be rather more than half the size of *F. nocturna* and *F. diurna*. It is sharp-tailed like these, but, in addition to the size, it differs from them inasmuch as it observes no periodicity, being present in the peripheral circulation both by day and by night, and, also, in not being enclosed in a sheath. Nothing is known of its life-history, minute anatomy, or pathological bearings. Possibly it is the embryonic form of *F. magalhæsi*—also a tropical American blood parasite.

I have recently met with, apparently, the same parasite in the blood of natives of St. Lucia, W.I., where Dr. Galgey has still more recently shown that

either it, or a similar blood worm, is very common. It is quite possible that the sharp-tailed filaria (*F. ozzardi*) of British Guiana is the same species.

I have also found a minute, non-sheathed, sharp-tailed embryo filaria in the blood of natives of New Guinea, likewise closely resembling *F. demarquati*. Whether these various embryos belong to one or to several species it is impossible to decide until the parental forms of each have been discovered and compared.

FILARIA OZZARDI. (Fig. 51, d.)

Some time ago I received from Dr. Ozzard, of the Colonial Medical Service, British Guiana, a number of blood films prepared from aboriginal Carib Indians inhabiting the back-country of that colony. Although the negroes and other inhabitants of the littoral and settled districts of British Guiana are very subject to *F. nocturna* and to elephantiasis, in none of the considerable number of slides of Carib blood from time to time received from Dr. Ozzard and Dr. Daniels have I once encountered *F. nocturna*. Dr. Daniels records an identical experience. I am assured by Dr. Ozzard that elephantiasis is unknown amongst these people. On examining the blood slides referred to, I discovered certain nematode embryos with characters so peculiar that I suspected they represented at least one new species of blood worm, which I called, provisionally, *F. ozzardi*. At least half of the slides examined contained these parasites, some slides only one or two, other slides as many as forty or fifty. In size and shape five out of six of the embryos resembled very closely *F. perstans* (p. 536)—that is to say, they were blunt-tailed, had no sheath, and were very minute (.173 to .240 mm. by .0043 to .005 mm., Daniels). But along with the blunt-tailed filariae and on the same slides there occurred a sharp-tailed form, also very minute, and resembling *F. demarquati*. There were about five blunt-tailed parasites to every sharp-tailed parasite. Drs. Ozzard and

Daniels (*Brit. Guiana Med. Ann.*, 1897) confirmed this discovery. Both of these observers have had abundant opportunity of examining these filariæ alive. They found that the sharp-tailed and the blunt-tailed worms behave in fresh blood on the microscope slide much in the same way that *F. perstans* (p. 537) does; that is, they wriggle about very actively, at the same time retracting, elongating, and locomoting in the blood.

Parental form.—For a time the relationship of these embryo filariæ, both to each other and to *F. demarquaii*, remained undetermined. Recently Dr. Daniels found parental filariæ—male and female—at the *post-mortem* examination of two Demerara Indians whose blood, during life, contained both blunt- and sharp-tailed embryos. The mature worms, apparently

TABLE.

	Filaria bancrofti.	Filaria perstans.	Filaria ozzardi.
	Mm.	Mm.	Mm.
Length	85 to 90	70 to 80	81
Greatest thickness ...	0·20 to 0·26	0·120	0·210
Diameter of head ...	0·055	0·070	0·050
Diameter of neck ...	0·049	0·054	0·039
Distance from head—			
(1) Of vaginal outlet ...	0·710	0·600	0·710
(2) Of ovarian opening ...	0·920	?	0·850
Distance from tail of anal papilla	0·225	0·145	0·230
Termination of tail ...	Blunt, circular, not bulbous.	Slightly bulbous; covered by thickened cuticle prolonged into two triangular appendages.	Bulbous cuticle not thickened.

numerous, were about three inches in length and very slender—about one-third the diameter of *F. bancrofti*. They were, therefore, not *F. mayalhæsi* (p. 538). The head was somewhat club-shaped and showed no papillæ. The tail of the male was much coiled and carried at least one long protruding spicule. These

worms were found in one case in the mesentery and in the fat at the base of the mesentery; in the other "not only in mesentery and abdominal fat, but also in the subpericardial fat." The embryos *in utero*, Dr. Daniels stated, were all blunt-tailed. I had an opportunity of comparing these worms with unquestionable parental *F. perstans*. I found them to be identical. The peculiar bifid arrangement of the termination of the tail was quite characteristic of that parasite (p. 537).

Later, Dr. Daniels (*Brit. Med. Journ.*, June 17th, 1899) found at the *post-mortem* examination of a third aboriginal in whose blood both the blunt-tailed (*F. perstans*) and the sharp-tailed filariæ had been found (and no others) a few adult *F. perstans* and, in addition, a female and portion of a male worm of quite a different species—presumably the parental form of the sharp-tailed embryo. The two latter parental worms lay close together and were believed to have been located in the sub-peritoneal connective tissues in the anterior abdominal wall. Except in the matter of the caudal extremity, which was bulbous, in size and structure they closely resembled *F. bancrofti*. Dr. Daniels has drawn up the table (on p. 535) of the leading dimensions of the three species—*F. bancrofti*, *F. perstans*, and of this possibly new filaria for which I propose to retain provisionally the name *F. ozzardi*.

FILARIA PERSTANS. (Fig. 51, e.)

Geographical range.—This parasite is very common in the blood of the natives of large districts in West Africa. I have found it in natives from Old Calabar and from the basin of the Congo—both in the coast negroes and in those from the interior. Daniels informs me that he has found it in a native of British Central Africa residing on the east side of Lake Nyassa. In many parts of the endemic districts it occurs in about half the population. Prof. Firket, of Liège, has confirmed this observation as

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regards the Congo district. Sometimes it occurs along with *F. diurna* and *F. nocturna* in the same individual. I have never found it in West Indian negroes, nor, in fact, in natives of any country except West tropical Africa, and in the aborigines of Demerara. I have twice found it in Europeans who had resided on the Congo.

Its characters.—*Filaria perstans* observes no periodicity, being present in the blood both by day and by night. In this respect it resembles *F. demarquaii* and *F. ozzardi*; and, like these, in size, shape, and anatomical detail differs very materially from *F. nocturna* and *F. diurna*.

The embryo in the blood measures, on an average, $\frac{1}{125}$ inch in length by $\frac{1}{5500}$ inch in breadth; but, as it possesses to a remarkable degree the power to elongate and to shorten itself, these measurements do not always apply. On the whole, however, it is manifestly much smaller than *F. nocturna* and *F. diurna*. It is further distinguished from these filariæ by the entire absence of a sheath, and by the characters of its caudal end, which is invariably truncated and abruptly rounded off. The taper terminating in the tail extends through quite two-thirds of the entire length of the animal. Further, if the head be carefully observed with a high power, a fang can generally be easily seen—much more easily than the corresponding structure in *F. nocturna*—in constant play, shot out and retracted. From indications in stained specimens I judge that it possesses a V- and a tail-spot; this point, however, I have not carefully studied. No hooked cephalic prepuce can be made out. Its movements also differ from those of *F. nocturna*, for it not only wriggles about, just as that parasite does, but it indulges from time to time in long excursions through the blood, moving freely all over the slide, locomoting in fact very much in the same way as *F. nocturna* does in the mosquito's stomach after it has cast its sheath.

Parental form.—The parental form, discovered by

Daniels in Demerara Indians, and subsequently identified by myself as that of *F. perstans*, like *F. bancrofti*, is a long, cylindrical, filiform nematode of the dimensions given in the foregoing table (p. 535). The body is smooth, without markings, the mouth simple and unarmed. The tail in both sexes is peculiar and characteristic; it is incurvated, and the



Fig. 73.—Tail of adult *Filaria perstans*.

chitinous covering at the extreme tip is split up as it were into two minute triangular appendages, giving it a mitred appearance (Fig. 73). The male is smaller than the female, but the specimens hitherto obtained did not permit of accurate observation on spicules and papillæ. It is open to question if the latter are present. So far the parental forms have been found in the connective

tissues at the root of the mesentery, behind the abdominal aorta, and beneath the pericardium.

Pathology.—What pathological rôle *F. perstans* may play is still uncertain. I have conjectured, for reasons stated elsewhere (p. 286), that it may in some way be the cause of negro lethargy and of one of the forms of African craw-craw (p. 563).

FILARIA MAGALHÆSI.

Professor Magalhães describes (*Rev. des Cursos theoreticos e prat. da Fac. de Med. de R. de Janeiro*, No. 3, An. III., 1886) two sexually mature filarial

hæmatozoa, male and female, which were found lying in the left ventricle of the heart of a child that died in Rio de Janeiro. No information was received as to the nature of the disease of which the child died, nor had any examination of the blood been made during life. The parasites were cylindrical, capillary, opalescent, white, uniform in thickness except where the body tapered towards the tail and at the club-shaped oral end. The mouth was simple, circular, unarmed; the cuticle marked with fine transparent striations. The female worm measured 155 mm. in length by 0·7 mm. in diameter, the male 83 mm. in length by 0·4 mm. in diameter. The tail of the latter was provided with four pairs of pre-anal and four pairs of post-anal papillæ, and two spicules. Manifestly this parasite is specifically distinct from *F. bancrofti*. The fact that it occurred in the Western hemisphere would suggest the possibility that it may be the parental form either of *F. demarquaii* or of *F. ozzardi*. Nothing is known of its life-history, nor of the associated pathology.

CHAPTER XXXIV.

ENDEMIC HÆMATURIA (BILHARZIA DISEASE).

Definition.—A form of hæmaturia caused by *Bilharzia hæmatobia* (*Distomum hæmatobium*), and characterised by the presence of the ova of that parasite in the urine.

History and geographical distribution.—The frequency of hæmaturia in the natives of Egypt, and in visitors to that country, has long been remarked. The explanation of this peculiar circumstance was supplied by Bilharz who, in 1851, discovered the cause in a peculiar trematode, subsequently named by Cobbold, in honour of its discoverer, *Bilharzia hæmatobia*.

So far as known, with the exception of Mesopotamia (Sturrock, *Brit. Med. Journ.*, Dec. 2nd, 1899), and possibly of Arabia, this parasite is limited to Africa and its island dependencies. Dr. John Harley discovered, in 1864, the characteristic ova in cases of hæmaturia from Natal. It has many times been found in patients from Mauritius. It has been found on the Gold Coast, in Tunis, and it probably exists in many other parts of Africa, more particularly along the eastern side of the continent. Egypt, however, is its favourite haunt; there, judging from Bilharz's, Griesinger's, and Sonsino's *post-mortem* records, it is present in quite one-half of the population.

Ætiology.—*The parasite* (Fig. 74).—*Bilharzia hæmatobia* belongs to the *Distomida*; but, unlike the majority of distomes, which are nearly all of them hermaphrodite, bilharzia presents the peculiarity of being bisexual. The *male* is white, cylindroid, measuring

15 mm. in length by 1 mm. in breadth. It possesses an oral and a ventral sucker placed close together. The cylindrical appearance of the worm is produced by the ventral infolding of the two sides of what would otherwise be a flat body. By this infolding a gynæcophoric canal is formed, in which the female can be partially enclosed. The female is rather darker in colour than the male, considerably longer (20mm.), more filiform, her middle being embraced during coitus by the gynæcophoric canal referred to, whilst her anterior and posterior portions remain free. The genital openings of the sexes face each other, and are placed immediately posterior to the ventral sucker.

These parasites are found in the blood of the portal vein, in its mesenteric and splenic branches, and in the vesical, uterine, and hæmorrhoidal veins. They have also been found in the vena cava; Sonsino considers that, if searched for, they would probably be found elsewhere in the circulation. Their numbers vary considerably. Sonsino reports finding in one case forty; in another case Kartulis found 300 in the portal vein and its branches.

The ovum (Fig. 75).—On microscopical examination the uterus of the female bilharzia is found to be stuffed with ova of a peculiar and characteristic shape. They are oval, each egg on an average measuring about 0.16 mm. in length by 0.06 mm. in breadth, one end of the ovum being provided with a short, stout,



Fig. 74.—*Bilharzia hematobia*, male and female, the latter in the gynæcophoric canal of the former. (After Leuckart.)

and very definite spine. In certain instances, hereafter to be mentioned, this spine is not terminal but is placed laterally.

The exact nature of the process by which the ova leave the body of the human host has not been satisfactorily explained. Apparently, the female worm migrates from time to time from the larger veins to their smaller radicles, and in these deposits her ova. The walls of the bladder and rectum are the favourite situations for this purpose. Afterwards the eggs are somehow carried, possibly



Fig. 75.—Ova of Bilharzia.

aided by the spine with which they are provided, towards the surface of the mucous membrane, and then, falling into the lumen of the bowel or into the bladder, are voided in the fæces or urine, a certain amount of blood escaping at the same time.

The free embryo (Fig. 76).—In newly voided urine the ovum presents a somewhat brownish appearance,

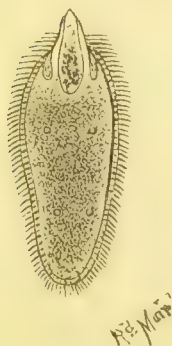


Fig. 76.—Free embryo of Bilharzia.

and generally contains a ciliated embryo. After a time the embryo may escape through a longitudinal rupture in the shell. It then swims about; but, unless supplied with fresh water, soon perishes. If the urine be freely diluted with water, the embryo not only escapes more quickly from the shell but also continues to live, swimming and gyrating very actively for a considerable time. While swimming, the body of the little animal undergoes many changes of shape.

For the most part, when advancing, it is oblong, tapering somewhat posteriorly; when more stationary it tends to assume a spherical form. It moves by means of the cilia which, with the exception of the minute papillary beak, thickly cover the entire body. On carefully examining the em-

bryo, a canal may be traced from the beak into what looks like a rudimentary stomach; on both sides of this two much smaller gland-like organs can be seen, from each of which a delicate tube passes forward and opens, apparently, somewhere in the neighbourhood of the beak. The bulk of the embryo is occupied by a number of sarcode globules. A careful description of the embryo is given by Dr. G. S. Brock in the *Lancet* of Sept. 9th, 1893, p. 625, to which the reader is referred for further details.

Life-history.—Beyond its first stage of free-swimming ciliated embryo the extracorporeal life of bilharzia is quite unknown, notwithstanding the many attempts that have been made to trace its future progress. Sorsino believed at one time that he had discovered its intermediate host in a fresh-water arthropod; this view he afterwards abandoned. Judging from analogy, the embryo bilharzia probably passes into the body of some fresh-water mollusc, into some crustacean, or into some larval arthropod; there, it is conjectured, it undergoes the developmental changes into redia and cercaria usually exhibited by the distomes. Later, it probably becomes encysted and then, either free or still in the body of the intermediate host, it gains access to man in drinking water and so, through the stomach, passes to the veins of the portal system.

Symptoms.—The symptoms produced by bilharzia vary in degree within very wide limits. Sometimes the patient experiences no trouble whatever; in other instances the suffering is very great. Indirectly, from the serious nature of the lesions of the urinary organs to which it may give rise, bilharzia is not an infrequent cause of death.

The most characteristic symptom of its presence in the wall of the bladder is the passage of blood at the end of micturition, with or without a sense of urinary irritation. The amount of blood so passed varies from a few drops of slightly tinged urine, to a considerable quantity of pure blood. The quantity of

blood passed and the degree of irritation are increased by exercise, by dietetic indiscretions, and by all such causes as are calculated to aggravate cystitis. As a rule, it is only the last few drops of urine that contain blood; sometimes, however, the hæmorrhage is more extensive, and then the entire bulk of the urine may be blood tinged. Occasionally, clots even are passed.

If in a case of moderate bilharzia infection the urine be passed into a glass and held up to the light, minute flocculi, or coiled-up mucoid-looking threads will be seen floating about in the fluid. If it be allowed to stand, the flocculi, and perhaps minute clots, will subside to the bottom of the vessel; these, on being taken up with a pipette and placed under the microscope, will be found to contain, besides blood corpuscles and the usual catarrhal products, large numbers of the characteristic spined ova.

In doubtful cases, where ova are few, the best way to find them is to get the patient to empty the bladder and to catch in a watch-glass the last few drops of urine which can be forced out by straining; these invariably contain ova. A low power of the microscope suffices and is best for diagnosis.

Endemic hæmaturia lasts for months or years. Recovery is rarely complete. In ordinary cases, provided no reinfection take place, the hæmaturia tends to decrease, although ova may continue for years to be found in the last few drops of urine passed. In severe cases, sooner or later, signs of cystitis supervene and give rise to a great deal of suffering. Not infrequently the ova become the nuclei for stone, and then symptoms of urinary calculus are superadded. Sometimes the pathological changes induced by the presence of the parasite in the bladder lead to the development of new growth, in which event the symptoms become more urgent and the hæmaturia perhaps excessive. Hypertrophy, contraction, or even dilatation of the bladder, are not unusual. Besides the bladder symptoms there may be signs of prostatic disease, or of disease of the vesiculæ seminales causing





spermatorrhœa. In the latter case, ova may be detected in the semen. In other instances the ureters and kidneys become involved, and grave disease of these organs ultimately ensues. In consequence of the suffering which these aggravated forms of infection produce, the patients become anæmic, wasted, debilitated, and a ready prey to intercurrent disease.

When the rectum is involved dysenteric-like symptoms may supervene, mucus with blood being passed from time to time, the stools becoming frequent and their passage being attended with tenesmus. In such cases small, soft growths are to be felt inside the sphincter ani. On removing one of these and breaking it up with needles, the spined ova can be readily made out in the *débris*.

In the female, vaginitis and cervicitis have been known to be produced by this parasite. The ova have also been found in the liver and in the lungs; so far as known, their presence in the latter organs gives rise to no active symptoms.

Pathological anatomy.—The character of the changes brought about by bilharzia varies very much according to the degree and the duration of the infection. In almost every case the walls of the urinary bladder are early affected. All that may be apparent to the naked eye at this stage of the disease is a certain amount of injection of the small vessels of the mucosa vesicæ, and, according to Sonsino, certain exceedingly minute vesicular or papular elevations of the surface of this membrane. When these minute elevations are examined microscopically they are found to contain ova. Ova are also to be found in the dilated minute blood-vessels. Later, especially in the trigone of the bladder, there are found rounded patches of inflammatory thickening which project somewhat, are granular on the surface, and dense in consistence; on section they creak under the knife as if they contained gritty particles. It is evident that these elevated, thickened patches are the result of an inflammatory process provoked by the clusters of ova

which the microscope reveals scattered throughout their entire extent. The ova are principally deposited in the submucosa, less extensively in the mucous membrane itself, still less abundantly in the muscular walls of the organ or in its subserous connective tissue. They tend to occur in groups, each of which is invested by a sort of connective-tissue capsule; or they may be lying in small blood-vessels which they occlude. Some ova are seen to have undergone calcification; others are still fresh, either segmenting, or already containing a ciliated embryo. On the surface of the rounded patches already mentioned, phosphatic deposits, also containing ova, are not uncommon; sometimes the patches present minute sloughs. In addition to these indurated patches, various forms of polypoid excrescence—sometimes ulcerated—may protrude from the mucous surface into the cavity of the bladder. These various hyperplasiæ frequently contain the adult parasite as well as ova.

In addition to what may be called the specific changes in the mucosa, the muscular coats of the bladder are generally hypertrophied. In consequence of this, as well as of the ingrowth of villousities and different forms of new growth, the capacity of the organ may be much diminished. Its mucous surface is generally coated with a sanguineous mucus containing myriads of ova. Gravel, or small stones are sometimes found either embedded in lacunæ in the hypertrophied and roughened bladder wall, or free in the cavity. Not infrequently a similar hyperplasia occurs in the ureters, particularly towards their lower ends. In rare instances the pelvis of the kidney itself is affected. Obliteration of the ureter, both from small stones and, also, from thickening of the mucous membrane, has sometimes been met with; this leads to dilatation of the pelvis and atrophy of the parenchyma of the kidney. It is easy to understand how, in time, these changes of the bladder and ureters may give rise to hydronephrosis, pyelitis, abscess of the kidney, and similar secondary affections.

Hyperplasia from bilharzia infection may also occur in the vesiculæ seminales, in the walls of the vagina, and in the cervix of the uterus, leading to corresponding bloody, ova-containing discharges.

In bilharzia disease, as regards practical importance, next to the bladder and consequent kidney affections rank the changes occurring in the large intestine, particularly in the lower part of the rectum. In this situation polypoid growths, apt to be mistaken for piles, are frequent. Large extravasations of blood under the mucous coat may also occur.

Strange to say, in ova found in the bowel, according to Soursino and Mackie, the spine is generally placed laterally; whereas in ova coming from the walls of the bladder the spine is invariably terminal. This lateral situation of the spine has been explained in various ways, the most probable suggestion being that it is a consequence of the compression exercised by the muscular coat of the intestine on the parent worm while the ova are passing the shell glands; the relations of certain of the reproductive organs of the worm are supposed to be altered by this compression.

It may be mentioned that ova in small numbers have been found in the liver, in gall-stones, in the lungs, in the heart, and in the kidneys. We have no knowledge of any definite pathological change entailed by their presence there.

Diagnosis.—The diagnosis of bilharzia disease is easy; the presence of ova in the urine is decisive. In countries like Egypt, where the disease must often concur with chyluria, with stone, with vesical tumour, with gonorrhœal cystitis, and with pyelitis, as well as with prostatic disease, care must be exercised in each particular case to separate the special factors to which the different symptoms are attributable. Thus in chyluria concurring with bilharzia disease there will be chyle in the urine in addition to blood. In such a combination the clot which forms will be large, will contain oil granules and globules and, very probably,

filaria embryos in addition to bilharzia ova ; moreover, the filaria will generally be detectable in the finger blood if looked for at night. Stone in the bladder, when suspected, has to be searched for with the sound. In gonorrhœal cystitis the history of gonorrhœa will be forthcoming. In prostatic disease, enlargement of this organ may be made out. Difficulty may sometimes arise when ova are few in number, or when they have ceased altogether to come away in consequence of the death of the parent worms. The mischief wrought by the parasite remains, although the ova—the most certain evidence of the parasite's previous presence—may be discharged no longer. But, even if ova are very few, they may still be found in the last drop or two of urine passed. If they are no longer to be found in the urine, sometimes, by scratching the surface of the bladder with a sound and examining the shreds of mucus so obtained, a few, calcified it may be, but presenting the characteristic spine, may be seen with the microscope. In rectal disease, if bilharzia be suspected, the mucus and the fæces, or, failing these, one of the polypoid growths after removal by finger or forceps, as well as the urine, should be examined for ova.

Prognosis.—An important element to be considered in venturing on a prognosis is the long life of the parasite. Sonsino mentions a case in which living ova were still being passed nine years after their first appearance, and after all chance of reinfection had ceased. Another important element in prognosis is the degree of infection ; the greater the number of worms the more severe and the more extensive the disease they produce. As with filarial infection, the greater the number of cases in a district the greater will be the probability of severe infections being met with. The prognosis is practically that of a chronic cystitis depending on an irremediable, but not in itself fatal, cause. Much suffering may often be looked for and, as a consequence, anæmia and debility ; possibly calculus may be formed ; possibly

grave renal disease may ensue. In the milder degrees of infection, which fortunately are the commonest, the patient seems to be in no way inconvenienced by the parasite, and generally escapes all serious consequences. In any case, mild or severe, there may be attacks of hæmaturia from time to time ; as a rule, the quantity of blood lost is insignificant.

Treatment.—Our knowledge of the situations occupied by the parasite indicates the futility of attempting a radical cure by means of poisonous substances, whether introduced by the bladder, by the rectum, or by the stomach. As yet we know of no direct, or other, means by which the bilharzia can be destroyed. Harm only can result from attempts at a radical cure of endemic hæmaturia on such lines. Our efforts must, therefore, be confined to palliating the effects of the presence of the parasite. Practically, the treatment resolves itself into that of chronic cystitis. The diet should be bland but nutritious ; stimulants and spices are to be avoided. Excess of all kinds, violent muscular efforts, cold and other causes of catarrh must also be guarded against. During exacerbations of hæmaturia, or of cystitis, rest should be enjoined and diluents freely partaken of. Pain may demand anodynes. Excessive catarrh of the bladder suggests washing out with weak boric acid lotion, and the internal administration of uva ursi, buchu, perhaps small doses of cubebs, copaiba, or sandalwood oil, salol, benzoic acid, and so forth. Stone, and troublesome new growths, are to be removed by operation. Where distress was extreme, Mackie had good results from cystotomy. Polypoid growths in the rectum, where accessible, should be removed. Hyperplasia in the vagina and cervix is best treated by scraping.

Prevention.—Since analogy justifies the belief that the embryo of bilharzia, on obtaining access to fresh water, enters a fresh-water animal and by it obtains access to another human host, it is evident that if the embryo be kept from getting into the

water, or, if drinking water be boiled or filtered, the spread of the disease from man to man would be effectually prevented.

In the endemic districts, children, in particular, should be carefully and repeatedly warned against drinking the water of ponds and canals. Provided reinfection be avoided by the exercise of prudence in the matter of drinking water, there is no necessity for sending the patient with bilharzia disease away from the country in which the parasite was acquired.





CHAPTER XXXV.

II. PARASITES OF THE CONNECTIVE TISSUE.

GUINEA WORM (*FILARIA MEDINENSIS*).

Geographical distribution.— This important parasite is found in certain parts of India—the Deccan, Scinde, etc.—in Persia, Turkestan, Arabia, tropical Africa—particularly on the west coast, and in a very limited part of Brazil (Feira de Santa Anna). Formerly it was supposed to be endemic in Curaçoa, Demerara, and Surinam; apparently it has now disappeared from these places. Guinea worm is not equally diffused throughout this extensive area; it tends to special prevalence in limited districts, in some of which it is excessively common. In parts of the Deccan, for example, at certain seasons of the year, nearly half the population is affected; and in places on the West Coast of Africa nearly every negro has one or more specimens about him. Although guinea worm is sometimes seen in Europe, this is only in natives of, or in recent visitors from, the endemic area. Though frequently introduced in this way it has not become acclimatised either in Europe or in North America. We have no account of the parasite as endemic in any part of Asia east of Hindustan, in the Eastern Archipelago, Australia, or in the Pacific Islands.

Guinea worm occurs occasionally in the lower animals—horse, dog, ox, etc. Possibly, some of the parasites in the lower animals described as guinea worm may belong to quite a different species.

The parasite (Fig. 77).—*Anatomical features.*—The female guinea worm alone is known; the male has not been recognised with certainty. She

is reputed to attain in some instances enormous dimensions; it is probable, however, that worms of



Fig. 77.—Guinea worm. (Reduced.) (Leuckart.)

five or six feet in length owe their size to errors of observation, two worms, or their fragments, having been regarded as one. According to Ewart, in forty carefully measured specimens, the smallest was $12\frac{3}{4}$ inches, the largest 40 inches in length; 30 inches is probably an average length. The diameter of the worm is about one-tenth of an inch. The body is cylindrical, milky-white, smooth, and without markings. The tip of the tail is abruptly bent, forming a sort of blunted hook, perhaps functioning as a "holdfast." The head end is rounded off, terminating in what is known as the cephalic shield. The mouth is triangular, very small, and surrounded by six papillæ—two large and four small. The alimentary canal is relatively small, being compressed and thrust to one side by the uterus; it is probably cæcal, for it has not been traced to an anus. The vagina, which must have existed at an early stage, at maturity has also become obliterated by the growth of the enormous uterus which, filled with coiled-up embryos (Fig. 78), extends from the head to the tail of the worm.

Habits.—The habitat of the female guinea worm is the connective tissue of the limbs and trunk.

When mature, in obedience to instinct, she proceeds to bore her way through this tissue, and, finally, travelling downwards, reaches a leg or foot. In 85 per cent. of cases she presents in some part of the



lower extremities ; occasionally in the scrotum ; rarely in the arms ; very exceptionally in other parts of the body, or even in the head. Arrived at her destination she pierces the derma. Probably in consequence of some irritating secretion, a small blister now forms and elevates the epidermis over the site of the hole in the derma. By-and-by the blister ruptures, disclosing a small superficial ulcer half to three-quarters of an inch in diameter. At the centre of the ulcer,



Fig. 78.—Section of guinea worm. (*Leuckart.*)

which sometimes quickly heals spontaneously, a minute hole, large enough to admit an ordinary probe, can be seen. Sometimes when the blister ruptures, the head of the worm is seen protruding from this hole ; as a rule, however, at first the worm does not show herself. If now we douche the neighbourhood of the ulcer with a stream of cold water expressed from a sponge and, as the water falls, watch the little hole in the centre of the ulcer, we shall see in a few seconds a droplet of fluid—at first clear, later milky—well up through the hole and flow over the ulcer. Sometimes, instead of this fluid, a small, beautifully pellucid tube, about $\frac{1}{16}$ of an

inch in diameter, doubtless the uterus of the worm prolapsed through her mouth, is projected through the hole in response to the stimulus of the cold



Fig. 79.—Embryos of guinea worm.
(From a photogram by Mr. H. B. Bristow.)

water. When this tube has been extruded an inch or thereabouts, it suddenly fills with an opaque whitish material, ruptures and collapses, the fluid spreading over the surface of the ulcer. If a little of the fluid, either that which has welled up through the hole, or that which has escaped from the ruptured

tube, is placed under the microscope, it is seen to contain myriads of embryo guinea worms lying coiled up, almost motionless, with their tails projecting in a very characteristic manner (Fig. 79). If now a drop of water be instilled below the cover-glass the embryos may be observed to unroll themselves and, in a very short time, to swim about, *more suo*, with great activity. Manifestly these embryos come from a guinea worm lying in the tissues and communicating with the surface through the little hole in the derma.

The embryo (Fig. 80).—The embryos are not cylindrical; they are distinctly flattened. In swimming they move by a sort of side-to-side lashing of the tail, and tadpole-like motion of the body. The movements are intermittent; sudden, short swims alternating with brief pauses. When progressing the longer transverse axis of the body is perpendicular to the plane travelled over; but when, from time to time, the little worms pause they gradually roll over on to their flat surfaces. As soon as they come to rest on the flat they suddenly recover themselves with a jerk, and turn quickly on to their edges and begin swimming about again. This series of movements is constantly repeated.

The embryo of the guinea worm measures about $\frac{1}{30}$ of an inch in length, by $\frac{1}{1000}$ of an inch at its greatest breadth. The head is

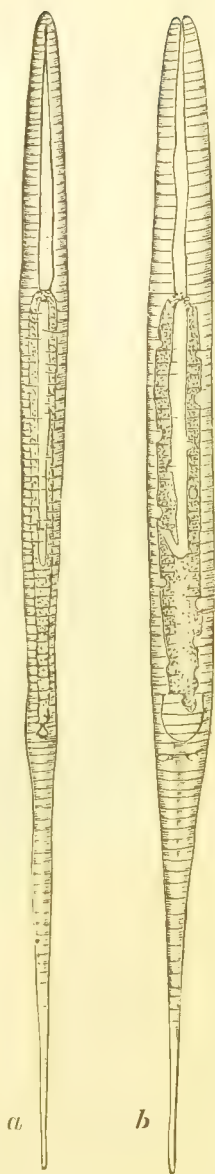


Fig. 80.—Embryos of guinea worm.

a, Side view; *b*, front view. ($\times 250$.)

somewhat tapered and then abruptly rounded off. The tail is long, slender, and sharp-pointed. The alimentary canal can be readily detected. Towards the root of the tail two peculiar gland-like organs, placed opposite to each other, can be made out. The cuticle is very distinctly transversely striated.

In clean water the embryo remains alive for six days; in muddy water, or in moist earth, it will

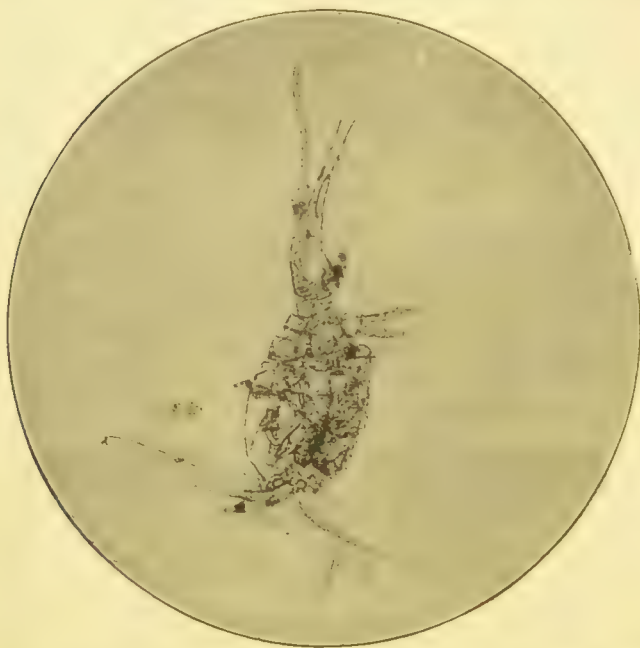


Fig. 81.—Embryos of guinea worm in body-cavity of cyclops. The cyclops has been slightly compressed so as to force out some of the worms, which can be seen escaping from ruptures at the head and tail. (From a photograph by Mr. Andrew Pringle.)

live from two to three weeks. If slowly desiccated it does not die; it may be resuscitated by placing it again in a little water.

Intermediary host.—If, by way of experiment, we place some guinea worm embryos along with *Cyclops quadricornis* in a watch-glass we shall find that, after a few hours, the embryos have transferred themselves

to the interior of the body-cavity of the cyclops, where they can be seen moving about, coiling and uncoiling themselves, with considerable activity (Fig. 81). As many as fifteen or twenty young guinea worms may be counted in each of the little crustaceans, which, unless the infection is excessive, seem in no way inconvenienced. After a time, the embryos so transferred undergo a metamorphosis. They cast their skins two or three times, get rid of their long swimming tails, acquire a cylindrical shape and, ultimately, along with increased size, develop a tripartite arrangement of the extreme posterior end, which recalls a similar arrangement in the tail of *F. nocturna* and of *F. recondita* towards the termination of the stay of the former nematode in the mosquito, and of the latter in the dog flea (Grassi).

Mode of infection.—The metamorphosis of guinea worm in cyclops was discovered by Fedschenko in Turkestan. His observations I have been able to confirm in England; but, in the colder climate of the latter country, in English cyclops the metamorphosis takes somewhat longer to complete; eight or nine weeks, instead of five weeks as in Turkestan. Fedschenko supposed that the cyclops containing the embryo guinea worm, on being swallowed by man in drinking water, was digested; and that the parasite, being then set free, worked its way into the tissues of its new and definitive host. It is possible, however, that the embryo may have to undergo further changes before it is fitted for life in man. Considering the peculiar geographical limitations of this helminthiasis, and the very general distribution of cyclops, such an arrangement as I suggest seems likely enough. However this may be, Fedschenko's discovery makes it almost certain that it is in drinking water that the guinea worm is acquired.

The male worm.—Nothing certain is known, as already stated, about the male worm; nor as to when or where impregnation of the female is effected. Impregnation probably occurs at an early stage of

parasitic life, and long before the female worm has grown to the great length which she subsequently attains. The male worm, his function fulfilled, probably dies and is absorbed.

Biological peculiarities explained.—The little we now know of the life-history of the guinea worm explains many things that were formerly mysterious. We now understand why the parasite, on attaining maturity, makes for the legs and feet; these are the parts of the human body most likely, in tropical countries, to come in contact with puddles of water, the medium in which cyclops—the intermediary host—lives. We can understand, also, why it is that the contact of water with the skin of the host causes the guinea worm to expel her young; and we can understand the rationale of the douching, so much practised by the natives of certain of the endemic districts, in their attempts at extraction.

Premature death of parasite.—Occasionally the guinea worm fails to pierce the integument of her host; sometimes she dies before arriving at maturity. In either case she may give rise to abscess; or she may become cretified, and in this condition may be felt, years afterwards, as a hard convoluted cord under the skin of the leg, or be discovered only on dissection.

Treatment.—Formerly it was the custom, so soon as a guinea worm showed itself, to attach the protruding part to a piece of wood and endeavour to wind her out by making a turn or two of this daily. Sometimes these attempts succeeded; but, just as often, the worm snapped under the strain. The consequences of this accident were often disastrous. Myriads of young escaped from the ruptured ends into the tissues, and violent inflammation and fever, followed by abscess and sloughing, ensued; weeks, or months, perhaps, elapsed before the unhappy victims of this rough surgery were able to get about. Too often serious contractions and ankyloses, from loss of tissue and inflammation and even death from septic trouble, resulted.



If a guinea worm be protected from injury, and the part she occupies frequently douched with water, her uterus will be gradually and naturally emptied of embryos. Until this process is completed she resists extraction; possibly the hook at the end of her tail assists her to maintain her hold. But when parturition, in from fifteen to twenty days, is completed, the worm is absorbed or tends to emerge spontaneously. A little traction if practised then may aid extrusion. Traction, however, must not be employed so long as the embryos are being emitted. The completion of parturition can be easily ascertained by the douching experiment already described.

Lately Emily, a French naval surgeon, has introduced a system of managing guinea worm cases which bids fair to shorten treatment and obviate the serious risks of the old winding-out system. By means of a Pravaz's syringe he injects the body of the worm, if she chances to be protruding, with solution of bichloride of mercury, 1 in 1,000. This kills the parasite; after twenty-four hours, extraction is generally easily effected. If the worm has not shown herself externally, but can be felt coiled up under the skin, he injects as near the coil as possible, and through several punctures, a few drops of the same solution. This, too, kills the parasite. Her body is then absorbed, as a piece of aseptic catgut would be, without inflammation or reaction of any description; or she may be cut down on, and easily extracted. Others have confirmed the value of Emily's method, which saves much time and suffering and, with due care, is devoid of risk.

FILARIA LOA (Fig. 82).

This parasite is peculiar to the West Coast of Africa where, in many parts, it is not uncommon. The male worm measures about 25 to 30 mm. in length by 0.3 mm. in breadth, the female 30 to



Fig. 82 *Filaria loa*. (Nat. size.)
(Dr. Argyll Robertson.)

40 mm. in length by 0.5 mm. in breadth. Both sexes are filiform, cylindrical, colourless, like fine fishing-gut, tapering slightly at the head, more decidedly at the tail. The body is dotted over with minute chitinous bosses. The anus is subterminal. The tail of the male (Fig. 83) is markedly incurvated and provided with five large papillæ on each side of the anus, and two rather short, unequal spicules. The mouth is simple, punctiform, and without armature.

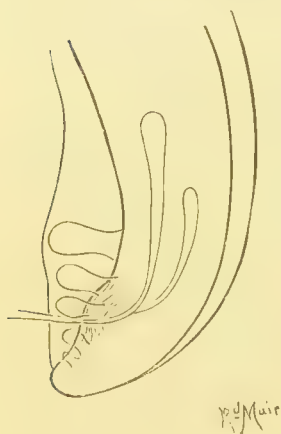


Fig. 83.—Tail of male *Filaria loa*.

The habitat of *Filaria loa* appears to be the connective tissue, which it traverses freely. When, in the course of its wanderings, it crosses the subconjunctival connective tissue, and the delicate integuments about the orbit, it becomes distinctly visible. When passing under such thin structures as the skin of the eyelids, or that over the bridge of the nose, or when moving about under the skin of the fingers and occasionally of other parts, the slight elevations it gives rise to, as well as its movements, are

sometimes appreciable, and may be both seen and felt.

From the circumstance that this parasite has occasionally been seen in individuals who have been ten or more years away from the endemic area in which alone it can be contracted, we may conclude that the loa is long-lived.

The female produces sheathed embryos closely resembling *F. nocturna* and *F. diurna*. At one time I had a negro patient under my care in whose blood *F. diurna* abounded. This man remembered that, when a lad, he had a loa in his eye. I have thought, therefore, that *F. diurna* may be the embryonic form of *F. loa*. On the other hand, Dr. Argyll Robertson has placed on record a remarkable case of *F. loa* in



which the parasites, a male and a pregnant female, were removed from the eye. The patient's blood was frequently examined, but no embryos were found. Lately I have had opportunities of examining the blood of other patients known to be the subject of loa infection, but failed to find in any of them hæmatozoal embryos. We do not know in what way the embryo escapes from the human body, nor do we know anything about its life-history outside of the human body. Notwithstanding the failures to find the embryo in the peripheral blood, analogy suggests that it probably circulates for a time in this fluid and, like *F. nocturna*, that it is removed from this medium by some suctorial insect which acts as intermediary host.

Filaria loa, as stated, is very active in wandering about the body, but it seems to have a predilection for the eyes and neighbourhood. It comes and goes there at irregular intervals of days, weeks, or months, traversing the subconjunctival tissue, and moving across the bridge of the nose from one eye to the other. Warmth seems to solicit its presence near the surface. The sexes appear to hunt each other about; at all events, in Dr. Argyll Robertson's case (*Trans. of the Ophthalmological Soc.*, 1895) a male worm was removed from the eye, and not long afterwards a female worm appeared about the same situation and was also removed. Blanchard (*Arch. de Parasit.*, October, 1899) has recorded a similar case.

The worm gives rise to pricking, itching, creeping sensations and, occasionally, transient oedematous swellings in different parts of the body. When it appears under the conjunctiva, it may cause a considerable amount of irritation and congestion. There may be actual pain even, associated with swelling and inability to use the eye and, perhaps, tumefaction of the eyelids.

Calabar swellings.—Under this name Mr. Thompstone describes (*Journ. of Trop. Med.*, Nov., 1899) certain fugitive swellings which, in the course

of six years' practice in Lower Nigeria he has encountered in three black and in four white men. The swellings, he says, are "about the size of half a goose egg, painless, though somewhat hot, both objectively and subjectively, not pitting on pressure, and usually disappear in three days. They come suddenly and disappear gradually, and occur in any part of the body." He never saw in the same individual more than one such swelling at a time, but they recur at irregular intervals even when the subject is in Europe. Plehn has seen similar swellings at Cameroon. Indeed, a lady, long resident in Old Calabar, informed me that if rubbing or scratching is not indulged in when a loa approaches the surface there will be no swelling, and that Calabar swellings are produced by the rubbing solicited by the irritation caused by *F. loa*. She herself had a loa extracted from under the skin over the right clavicle, and spoke from personal experience. Mr. Thompstone failed to find embryo filariæ in his patients. I had an opportunity of examining the blood of such a case recently and also failed to find hæmatozoa. The character of the swellings and the geographical range of this singular affection are not incompatible with filarial infection, possibly with *F. loa*.

Treatment.—The negroes treat loa in two ways—either by dropping a grain of common salt into the conjunctival sac, which has the effect of driving the filaria away, for a time at least; or they extract it with a sharp thorn, or by some other rude surgical means. With proper instruments there is no difficulty in removing the parasite from under the conjunctiva or from the eyelid—that is, if the operator is possessed of the requisite deftness in operating. All that is necessary, on the parasite showing itself, is, after cocainising the eye, to seize the conjunctiva with a pair of forceps, taking care to include the worm and the subconjunctival connective tissue in the grasp of the instrument. Having fixed



The purulating or vesiculating dermatitis of
the feet described by an anonymous writer
in the Journ. of Imp. Med., 15th Dec 1840, is caused by
~~some~~ the same cause as in the same is pro-
duced by the same nature as even even-
In also it is called Pani Shad. water
sore or sore foot.

the parasite in this way, the conjunctiva may be snipped through with scissors ; when, with the aid of another pair of forceps, and after releasing the first pair, the parasite may be withdrawn. In the case of the parasite showing itself elsewhere, I would suggest that it might be killed, as in the case of the guinea worm, by local hypodermic injection of bichloride of mercury solution (1 in 1,000).

FILARIA VOLVULUS.

A medical missionary sent Prof. Leuckart two tumours, the size of a pigeon's egg, which had been removed, one from the scalp, the other from the chest, of Gold Coast negroes. The tumours contained several male and female filariæ, the former 60 to 70 cm., the latter 30 to 35 cm. in length. The worms were coiled up in the form of a ball, and were bathed in a fluid full of embryos resembling *F. nocturna* and *F. diurna* ; no sheath, however, was visible. Nothing further is known of these parasites and the diseases, if any, to which they may give rise.

MM. Labadie-Lagrave and Deguy describe (*Arch. de Parasitologie*, July, 1899) a nematode worm found in a small tumour excised from the arm of a French soldier who had served in Dahomey some years previously. The worm occupied, apparently, a lymphatic vessel, and was coiled up and embedded in a tunnel in a circumscribed mass of connective tissue about the size of a pigeon's egg. It is difficult from the description to be sure that the authors are correct in assuming that the parasite in question was *F. volvulus*.

CRAW-CRAW.

Most itching papular and pustular eruptions are termed kra-kra by the natives of the West Coast of Africa. Dr. John O'Neil describes (*Lancet*, Feb., 1875) under this name a pustular affection which he says is common in certain parts of the West Coast,

and which he found to be associated with the presence of a filariform parasite in the papules. O'Neil says that this form of crawl-crawl resembles scabies; but he adds that symptoms subside in a cooler climate, to return, however, when the negro revisits the hot and damp atmosphere of his native country. The papules occur all over the limbs and body, either singly or in rings. In two days from its appearance the papule, he says, becomes a vesicle, and in two more a pustule.

On paring off the top of the papule with a sharp knife, and teasing up the little piece of integument in water, he found a number of minute filaria-like organisms wriggling about with great activity. Their activity speedily slowed down and in a short time the worms died. These organisms, according to O'Neil's drawings and description, resemble somewhat *F. nocturna*. The measurements, however, do not quite correspond, the crawl-crawl filaria being shorter and broader ($1\frac{1}{10}$ inch by $2\frac{1}{10}$ inch) than *F. nocturna*; moreover, unlike the latter, it presented two black markings at the cephalic end. He says that if the section of the papule be made sufficiently deep, five or six of these parasites may be seen in a field.

Crawl-crawl is said to be contagious. It appears after an incubation period of three days, and is not curable by sulphur inunction.

O'Neil's observations have not been confirmed. I think it is quite possible that the parasite he found was one of the several blood filariæ we now know to be so common on the West Coast of Africa. It is comprehensible that in a country in which *F. perstans* occurs in every second individual, it would be frequently found in such preparations as Dr. John O'Neil examined. The removal of the top of a scabies papule would certainly be attended with some degree of hæmorrhage; in which case, should the patient chance to be the subject of any form of filarial infection, these parasites would be found in the preparation. Immersion in water would, as in the case of O'Neil's parasite, quickly kill the parasites. I do not





wish to assert that O'Neil's parasite was *F. perstans*, but the possibility of this must not be overlooked.

A disease resembling O'Neil's crawl-crawl was described some time ago (*Archives de Méd.*, April, 1882) by Prof. Nielly under the title "dermatose parasitaire." A French lad, who had never been abroad, became affected with a papulo-vesicular itching eruption resembling scabies, in which Nielly found a filariform parasite somewhat like that discovered by O'Neil in crawl-crawl. It had the same peculiar cephalic markings; in addition, it had a well-defined alimentary canal and rudimentary organs of generation. Nielly found nematode embryos in the blood in this case; so that we are justified in believing that the parasite in the skin was an advanced developmental form of the embryo in the blood, and that both were the progeny of a mature parental worm living somewhere in the tissues. Possibly Nielly's dermatose parasitaire and O'Neil's crawl-crawl were of the same nature. I have suggested that, as the skin parasite in O'Neil's disease may have been an advanced form of *F. perstans*, this parasite normally, and in pursuance of its evolution, escapes from the human body through the skin after undergoing there a certain measure of developmental advance. Further investigations on this subject are much wanted.

Treatment.—The term crawl-crawl is very loosely applied. Emily (*Arch. de Méd. Navale*, January, 1899) has described under this name a papulopustular skin affection which is common in certain parts of tropical Africa, and which is often the cause of much suffering to the traveller. It, or a similar disease, is by no means confined to Africa, for I have seen it in patients from India and was at one time very familiar with it in South China. At the earliest stage the disease begins as an itching papule, very possibly at the seat of a mosquito bite. The itching provokes scratching, whereby some form of pyogenic micro-organism is inoculated. Pustulation follows, and is

spread over feet and legs by soiled shoes and stockings and auto-inoculation. In this way an ulcerating, pustulating dermatitis is kept up. Emily describes a very efficient treatment. Pustules are opened, crusts removed, and ulcers scraped. Boric acid powder is then dusted freely on the parts after a thorough scrubbing with sublimate lotion (1-1000), boricated vaseline applied on lint, and over all absorbent cotton and a bandage. The dressings are not disturbed for a week, when the parts will be found soundly healed. Such and similar auto-infective diseases of the hands and feet, so common in the tropics, I used to treat with a foot-bath of warm carbolic acid lotion (1-20), and dry dressing with abundance of boric acid powder, at the same time insisting on destruction of infected slippers, shoes, and stockings.



CHAPTER XXXVI.

III. PARASITES OF THE LUNGS.

ENDEMIC HÆMOPTYSIS: DISTOMUM RINGERI.

(*v. D. Westermanni, v. pulmonale.*)

Geographical distribution.—So far as known, this disease is confined to Japan, Corea, and Formosa. A doubtful case has been reported by Maxwell (*Journ. Trop. Med.*, December, 1899) in Changpoo, Fokien, China. In the endemic districts a notable percentage of the population is affected. It is not improbable that, as knowledge extends, the disease will be found to exist in other countries. Recently the parasite which gives rise to this peculiar form of blood-spitting has been found in the United States, both in the cat and in the dog. It is not at all unlikely, therefore, that ere long we may hear of endemic hæmoptysis from America. The Chinese and Japanese are nowadays to be found in almost every land, and doubtless they carry with them their peculiar parasites—*D. sinense*, *D. crassum*, and also *D. ringeri*.

Symptoms.—The subjects of endemic hæmoptysis have a chronic cough, which is usually most urgent in the morning on rising. The fits of coughing eventuate in the expulsion of a peculiar rusty brown, pneumonic-like sputum. This sputum can be produced at will almost at any time, and often in considerable quantity. In addition to the chronic cough and the rusty expectoration referred to, the patient is liable to irregular attacks of hæmoptysis. Though usually induced by violent exertion, occasionally such attacks come on without apparent cause. The hæmoptysis may be trifling; on the other hand,

it may be so profuse as to threaten life—at all events, to cause intense anæmia.

The sputum.—On placing a minute portion of the viscid, pneumonic-like sputum under the microscope, its peculiar colour is found to be due partly to red blood corpuscles, partly to a crowd of dark brown, thick-shelled, operculated ova (Fig. 84). These ova

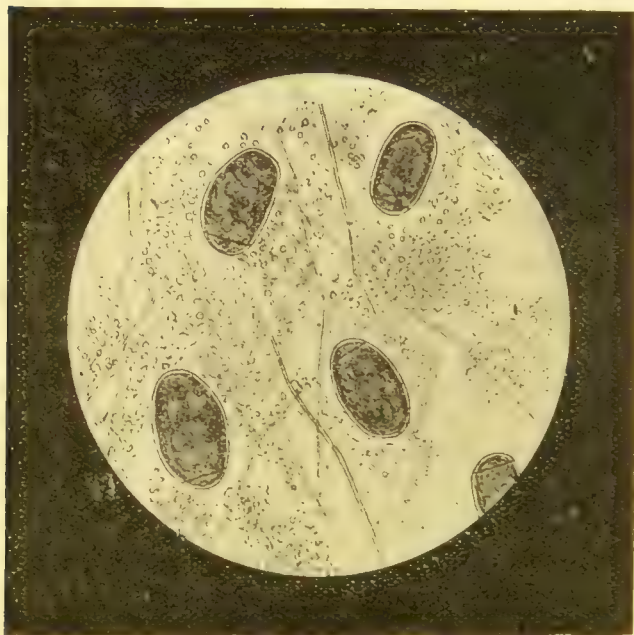


Fig. 84.—Ova of *Distomum ringeri* in sputum.

vary a good deal in size and shape; they are all distinctly oval, smooth, double-outlined, and measure from 80 to 100 μ in length, by 40 to 60 μ in breadth. If the sputum is shaken up in water, and the water be renewed from time to time, in the course of a month or six weeks—longer or shorter according to temperature—a ciliated embryo is developed in each ovum. When the ovum is mature, on placing it on a slide and exercising slight pressure on the cover-glass,

the operculum will be forced back, and the embryo will immediately emerge and at once begin to swim and gyrate in the water. Farther than this the life-history of the embryo has not been traced; but, doubtless, it is continued in some fresh-water animal through which it finds its way back, in a more or less direct way, to man.

Pathological anatomy.—On making a section of the lungs in this disease, a larger or smaller number of what are known as “burrows” are discovered scattered about this organ, particularly towards the periphery. These burrows consist of areas, somewhat larger than a filbert, of infiltrated lung tissue in which can be seen a number of tunnels filled with the same material that constitutes the characteristic sputum, and also containing one, two, or more small distomes. The septa between the tunnels may break down and a considerable cavity be thus produced; and as this occurs in connection with one of the bronchi, with which the tunnels always communicate, it may give rise to the appearance of a dilated bronchus. One burrow may communicate with another.

When first discovered it was supposed that *D. ringeri* (or *D. pulmonale*, Leuck.) was confined to the lungs. Later investigation has shown that it may affect the liver, peritoneum, testes, and even the brain. In the latter it forms a sort of tunnelled tumour similar to those in the lungs; and, by the pressure or irritation proceeding from this tumour, may give rise to a peculiar and ultimately fatal form of Jacksonian epilepsy.

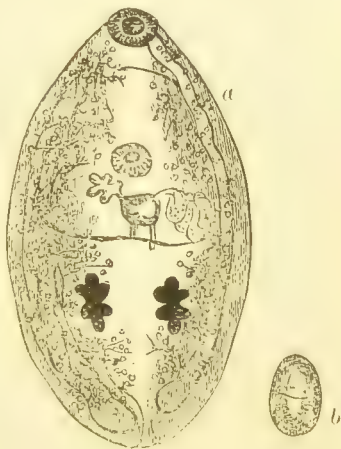


Fig. 85.—*Distomum ringeri*.
(Leuckart.
a, Magnified; b, natural size.

The parasite (Fig. 85).—The parasite itself is reddish brown in colour, thick and fleshy, and oval in form. So thick is it that its transverse section is almost round. It measures 8 to 10 mm. in length by 4 to 6 mm. in breadth, and is covered with minute spines.

Diagnosis.—Diagnosis of endemic hæmoptysis is at once established by the discovery of the characteristic ova in the almost equally characteristic sputum. Râles and other physical signs of lung consolidation are not usually discoverable.

In the case of one-sided convulsions, or in hemiplegic affections occurring in a native of, or in a visitor from, the countries in which this distome is endemic, the sputum should be examined on the chance of discovering evidence of the parasite. Should ova be found, there is a strong presumption that the cerebral trouble arises from distomum tumour in the brain.

Treatment.—Hitherto no means of expelling this parasite from the lungs has been discovered. In the case of cerebral distomiasis it might be possible by an operation to remove the parasite and associated tumour, and thus afford a chance of recovery in what has hitherto proved a fatal condition.

Prophylaxis in this, as in so many animal parasitic diseases, principally lies in the direction of securing a pure water-supply and avoiding all uncooked articles of diet which might be supposed to contain the young parasites.





CHAPTER XXXVII.

IV. PARASITES OF THE LIVER.

DISTOMUM CONJUNCTUM.

DISTOMUM CONJUNCTUM (Fig. 86, *a, c*) was discovered by MacConnell in an East Indian in 1874. It is leaf-shaped, narrower in front than behind, and measures 9 to 12 mm. in length by $2\frac{1}{2}$ mm. in breadth. Its surface is covered with minute spines. The eggs (Fig. 66, *b*) are oval, operculated, and measure $34\ \mu$ in length by $19\ \mu$ in breadth. This parasite inhabits the bile ducts, which it thickens and sacculates. Apparently *D. conjunctum* is but of slight importance pathologically.

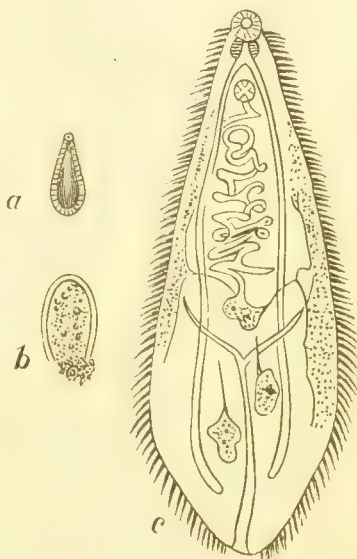


Fig. 86.—*Distomum conjunctum*.
a, Natural size; *b*, ovum; *c*, magnified.

DISTOMUM SINENSE.

Distomum sinense was discovered almost simultaneously by MacConnell in India and MacGregor in Mauritius, in 1874.

It has been found in many Eastern countries, including India, Mauritius, Japan, Corea, Formosa, China, and Tonkin. In the last-named country it appears to be very common.

D. sinense (Fig. 87, *a, c*) measures from 20 to 22 mm. in length; it is oblong, narrow, and some-

what pointed anteriorly, reddish in colour, and almost transparent. The eggs (Fig. 87, *b*) are 28 to 30 μ in length by 16 to 17 μ in breadth, operculated, almost



Fig. 87.—*Distomum sinense*
a, Natural size; *b*, ovum; *c*, magnified.

black in colour, and contain a ciliated embryo. Nothing is known of the life-history of the parasite farther than that it may occur in some of the lower animals—in the cat, for example. Probably its immature stages are passed in a mollusc or other small, soft-skinned, fresh water animal.





D. sinense inhabits the bile ducts and gall-bladder. It dilates and thickens the biliary canals, expanding them in places into cavities and diverticula as large as a filbert. In these cavities vast numbers of parasites are sometimes found. The diverticula communicate with the bile ducts, along which the ova of the parasites, and sometimes the parasites themselves, escape into the intestine. The affected liver is enlarged on the whole, although the tissue in the immediate neighbourhood of the diseased bile ducts is atrophied. The spleen, also, may be hypertrophied, and the intestine in a condition of chronic catarrh.

This parasite, which for long was supposed to be practically innocuous, is now known to be the cause of a serious disease of the liver which may terminate fatally.

Baelz discovered that in certain low-lying, unhygienic villages in Japan this helminthiasis was exceedingly common, quite 20 per cent. of the inhabitants being affected. When the infection is severe the liver becomes enlarged, and chronic diarrhœa, with recurring attacks of jaundice, sets in. Later, anasarca appears, and gradually a cachexia resembling that of sheep-rot is established which, in the course of several years, may prove fatal.

It would be well to bear this parasite in mind in approaching the diagnosis of obscure hepatic disease, associated with diarrhœa and jaundice, in patients from the East. It is just possible that the discovery of the ova in the stools might guide to a correct diagnosis.

PENTASTOMUM CONSTRICTUM (Fig. 88).

This, which is probably a formidable parasite, has now been found a number of times encysted in the liver, and occasionally in the lungs, of African negroes. It appears to give rise to a considerable amount of irritation, perhaps to peritonitis and to

pneumonia. Nothing is known as to the way in which man becomes infected with the parasite, of which the larval form alone has been recognised. This is of a milky white colour, from 1 to $1\frac{1}{2}$ inch in length, cylindrical, flattened on the ventral surface,

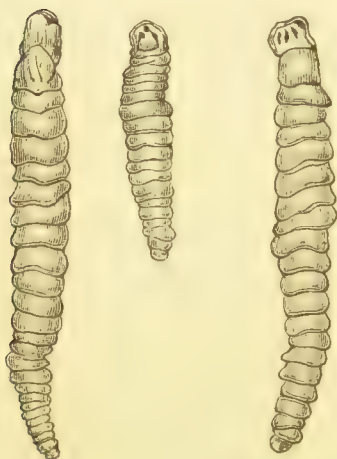


Fig. 88.—*Pentastomum constrictum*.
(Aitken.)

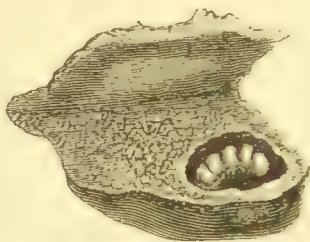


Fig. 89.—*Pentastomum constrictum* encysted in the liver.
(Aitken.)

ringed, rounded anteriorly, and terminating posteriorly in a blunt cone. The anterior end is provided with two pairs of hooks arranged on each side of the pit-like mouth. The posterior aspect of the rings, twenty-three in number, carries a number of minute spinous projections. The parasites, surrounded by their integumental exuviae, are found each coiled up in a cyst-like pouch, and scattered through the substance of the liver (Fig. 89), or projecting as nodules on the surface of the gland. Aitken ("Principles and Practice of Medicine," 4th ed., 1866) gives a very complete account of a case of a negro in whom the pentastomum seemed to be the cause of death. He gives a drawing from another case showing the cysts in the liver, from the surface of which one of the parasites is protruding into the peritoneal cavity.





CHAPTER XXXVIII.

V. INTESTINAL PARASITES.

NEMATODES.

Microscopical examination of the fæces for ova of intestinal parasites.—If the fæces of the natives of warm climates, and of Europeans coming from warm climates, are systematically examined with the microscope, it will be astonishing how frequently they are found to contain the ova of three species of nematode worms—*Ascaris lumbricoides*, *Trichocephalus dispar*, and *Ankylostomum duodenale*. The ova of the tapeworms and of the common threadworm (*Oxyuris vermicularis*), are rarely found in the stools, as these parasites do not, as a rule, part with their ova until the joints of the former, or the entire body of the latter, have left the alimentary canal; but as the three nematodes mentioned pass their eggs directly into the bowel, these eggs habitually appear in the fæces, and constitute unequivocal evidence of the presence of their respective parental forms. Occasionally the ova of the rarer hepatic and intestinal parasites—such as *Bilharzia hematobia*, *Distomum sinense*, *Distomum crassum*, *Distomum hepaticum*, and one or two still rarer helminths are encountered; as these, with the exception of *bilharzia*, are very rare, from a practical point of view they may be disregarded. Without large experience, the ova of the rarer parasites cannot be identified off-hand; but if the practitioner has learned to recognise those of the three common species, he will at once know when he comes across the ova of any of the rarer species and, on referring

to some special work on helminthology, will have little difficulty in arriving at a correct diagnosis.

The microscopic examination of fæces for ova, though somewhat disagreeable, is by no means a difficult matter. All that is necessary, by way of preparation, is to place on the slip a minute portion of the suspected fæces—about the size of a hemp seed—and then to apply the cover-glass, gently gliding it over the slip so as to spread out the mass in a thin, fairly uniform, and transparent layer. If the bit of fæces prove too consistent, a little water may be added so as to soften it. If the stools are loose and watery, the sediment should be taken up with a pipette and examined. It is well to make two or three preparations. The microscopic examination must, in the first instance, be made with a low power—an inch, or better, a half-inch objective. Search must be made in every part of the slide, and every suspicious body carefully scrutinised, a higher power being used if necessary. A very little practice suffices for the identification not only of ova but of the species of parasite to which the ova belong.

The points to be attended to in the diagnosis of ova are size, shape, colour, thickness, roughness, smoothness, and markings on the surface of the shell; the presence or otherwise of yolk spheres, of a differentiated embryo, or, in the case of the tæniæ, of the three pairs of embryonic hooklets; the existence of an operculum in the case of certain distomes and of bothriocephalus. The ova of the same species of parasite vary but slightly, and are in every instance sufficiently stable and definite for correct diagnosis.

Ova of Trichocephalus dispar (Fig. 90, a).—Of the three common nematodes mentioned—*Trichocephalus dispar*, *Ascaris lumbricoides*, and *Ankylostomum duodenale*—the ova of the first are those most frequently met with. They occur sometimes in enormous numbers, as many as six or eight specimens being visible in one field of an inch objective. They form rather a striking object under the microscope. They are oval, measuring





from $56\ \mu$ by $24\ \mu$ to $36\ \mu$ by $26\ \mu$, the ends of the long axis of the oval being slightly pointed and tipped with a little shining protection or plug. Their general appearance suggests an elongated oval tray, the projections at the poles of the ovum representing the handles of the tray. They are dark brown in colour, sharply defined, double outlined, and contain no differentiated embryo.

The ova of *Ascaris lumbricoides* (Fig. 90, b) are considerably larger (60 to $75\ \mu$ by 36 to $55\ \mu$) than those of *Trichocephalus dispar* (Fig. 90, a). They are



Fig. 90.—Ova of a, *Trichocephalus dispar*; b, *Ascaris lumbricoides*; c, *Ankylostomum duodenale*. (Sousino.)

also, as a rule, more spherical or, rather, more broadly oval; occasionally they are almost barrel-shaped. Like those of *trichocephalus*, they are dark brown in colour from bile staining, but they are much less sharply and smoothly defined, possessing a coarse thick shell which is roughened by many warty excrescences. The yolk contents are not always easily made out, nor, when made out, can any sign of embryo or segmentation be discovered.

A point of practical importance to be attended to lies in the circumstance that the rough outer layer on the shell of the ovum of *ascaris* is very easily detached, leaving the egg with a sharp, smooth outline suggestive of some other form of parasite. Therefore, to obviate this, in mounting faeces it is well to avoid too much gliding of the cover-glass over the slip.

The ova of *Ankylostomum duodenale* (Fig. 90, c) contrast very markedly with both the foregoing, particularly in the matter of colour. *Trichocephalus* and *ascaris* ova are invariably dark and bile-stained; those of the *ankylostomum* are beautifully clear and transparent. They measure 55μ to 65μ by 32μ to 43μ ; have a regular, somewhat elongated oval form, with a delicate, smooth, transparent shell, through which two or four light grey yolk segments can be distinctly seen. It is well to search for these ova soon after the fæces have been passed; otherwise, owing to the rapidity with which, in favourable circumstances, development proceeds, the embryo may have quitted the shell and the egg be no longer visible.

TRICHOCEPHALUS DISPAR.

Trichocephalus dispar, the whip-worm, lives principally in the cæcum. In many countries it is present in more than half the population. It is believed to maintain its position by transfixing, pin fashion, with its long slender neck a superficial fold of the mucous membrane. According to Powell, the females very much preponderate, the proportion to males being as 466 to 1. Excepting that the practitioner should be familiar with the appearance of its eggs in the stool, so that he may be able to distinguish them from those of *ascaris*, of *ankylostomum*, and of other parasites, its presence is of no practical moment. So far as known, it gives rise to no serious pathological lesion; a fortunate circumstance, seeing that hitherto it has been found impossible, with any degree of certainty, to dislodge it by anthelmintics.

ASCARIS LUMBRICOIDES.

Though not quite so common in tropical countries as *trichocephalus*, the *ascaris* is nevertheless very common indeed, especially in children, who often harbour these loathsome creatures in enormous numbers





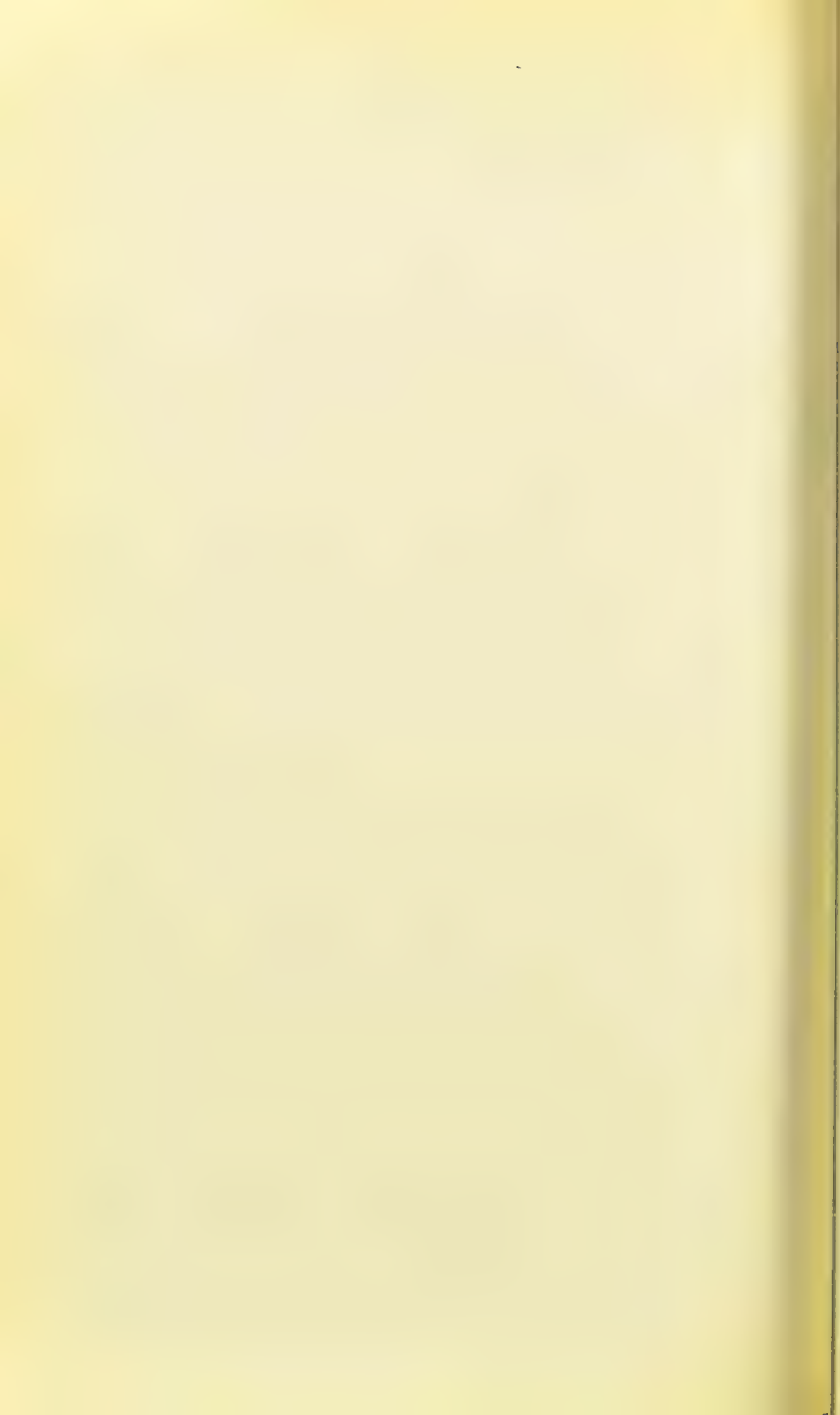
—in dozens, or even in hundreds. In those countries, at one time or another, nearly every child gets them ; so much so that, when doubt exists about the nature of some obscure affection, a dose or two of *santonin* often produces results which will seem to justify a diagnosis of “worms,” and, for the time being perhaps, satisfy an anxious mother.

Mode of infection.—The reason for the great frequency of *Ascaris lumbricoides* in tropical countries is probably twofold—the warmth of the climate, and the habits of the people with regard to the disposal of night-soil. In the *fæces* the ova exhibit no trace of segmentation or of differentiated embryo ; but if placed in water, or kept moist and in a warm place, in the course of from five or six months—longer or shorter according to temperature—the embryo is developed, and can be seen coiled up and moving about inside the egg-shell. If such an egg is accidentally or intentionally swallowed, on arrival in the stomach the shell is dissolved away and the contained embryo is set free. In a month it grows into a sexually mature animal, and, if both sexes are present, eggs in countless numbers are soon produced and appear in the *fæces*. Desiccation of the egg at atmospheric temperatures does not destroy the embryo, which will quickly revive on becoming moistened. In many warm countries night-soil is the favourite fertiliser, and is regularly preserved and spread upon the fields. In this way the ova of *ascaris* obtain an opportunity of maturing, and thus, too, they have an opportunity of being swallowed by man. They may also be washed into drinking water ; or, becoming desiccated on the drying up of the fields, be blown about as dust ; or they may become attached to fruit or vegetables. In one of these, or in similar ways, they finally reach the human stomach and there attain maturity.

Symptoms.—In many instances the *ascaris* gives rise to no very noticeable symptom ; in other instances it is to be credited with a number of ill-defined

gastric and perhaps nervous troubles—capricious appetite, foul breath, restless sleep, peevishness, vague abdominal pains, nausea, and so forth. Sometimes the worms get into the stomach and are vomited, their appearance giving rise to no inconsiderable alarm. They may even creep up the œsophagus and into the mouth, or out by the nostrils. Cases are on record in which they caused suffocation by wandering, in this way, into the rima glottidis. They have also been known to enter the bile ducts and give rise to jaundice; to penetrate the intestinal wall and escape into the peritoneum, causing peritonitis; or to burrow into the abdominal walls and cause abscess. Fortunately these accidents are of rare occurrence; their possibility, however, should be borne in mind and, apart from other obvious considerations, ought to make us endeavour to rid patients of these troublesome guests as soon as possible. With this object in view, it was my practice in China to give my little patients, as a matter of routine, a few doses of santonin twice a year; very often the precaution received its justification by the appearance in the stools of one or more ascarides.

Adults, especially young adults, although to a much smaller degree than children, are liable to entertain these verminous visitors. Sometimes certain obscure dyspeptic symptoms in grown-up people will resist all treatment until three or four grains of santonin and a purgative have been administered. I had a patient once, who for a long time had been troubled with unaccountable nausea. One day, while he was sitting at breakfast, the feeling of sickness came on with unusual intensity. He had to leave the table and, after one or two retching efforts, brought up an *Ascaris lumbricoides*. After this he was no more troubled with nausea. It is well, therefore, when puzzled over some obscure dyspeptic condition in tropical patients, to bear the ascaris in mind. If, for some reason, it is undesirable to give santonin unnecessarily, the stools ought first to be examined





with the microscope. If ova (Fig. 90, *b*) are found, a dose or two of santonin may clear up diagnosis and cure the patient; if no ova are found, the drug may be withheld and the idea of ascarides abandoned.

Treatment.—The ascaris is readily expelled by a few grains of santonin. The dose is from half a grain to one grain for a child, three to four grains for an adult. A good plan of giving the drug is to prescribe three such doses on successive nights, the first and last dose to be followed by castor oil next morning. Patients, or mothers, ought to be warned about the peculiar effect santonin has on the urine and sometimes on the vision. I have only once seen any bad effects; in this instance a peculiar sort of intoxication, attended with delirium, which did not quite pass away for several days, followed its exhibition.

ANKYLOSTOMUM DUODENALE AND ANKYLOSTOMIASIS.

Ascaris lumbricoides, though an unpleasant parasite, cannot be considered a dangerous one, unless in very exceptional circumstances. It is otherwise with the ankylostomum (sometimes called *Dochmius duodenalis*) which, in many countries, on account of the dangerous cachexia, called ankylostomiasis, it gives rise to, amounts to a positive curse.

Nomenclature.—The form of endemic anæmia with which this parasite is associated is of so marked a character that it has received a variety of distinctive names. Thus, in the French West Indies, severe ankylostomiasis is known as *cachexia aqueuse*; sometimes as *malcœur*, or as *mal d'estomac des negres*; in Colombia it is called *tuntun*, the sufferers being known as *tunientos*; in Brazil it has been called *oppilatio*, *opilação*, and *cangaço*; in Europe it is sometimes known as “miners’ anæmia,” or “tunnel disease,” the latter in allusion to the notorious Saint Gothard epidemic; the form occurring in Egypt is spoken of as Egyptian chlorosis; in Ceylon it has

been called beriberi; and, doubtless, elsewhere there are local names for this peculiar verminous anæmia.

Geographical distribution.—Since its discovery by Dubini in 1838, the ankylostomum has been found so widely diffused that it may be said to occur in all tropical and subtropical countries. Besides being abundantly present in the south of Europe, and in the tropical and subtropical regions of Asia and America, it has been ascertained to exist in North and South Queensland, Australia (*Trans. Intercolonial Med. Cong. of Australia*, 1893, Drs. Gibson and Turner), and in several of the islands of the Pacific. So prevalent is it in many parts of India that, according to Dobson (*Trans. First Ind. Med. Cong., Calcutta*, 1895), quite 75 per cent. of the inhabitants are affected. In Egypt it is found at nearly every *post-mortem* examination; and there the anæmia it gives rise to is one of the most common



Fig. 91.—*Ankylostomum duodenale*, male and female. (Blanchard.)

causes for the rejection of recruits in the army. Thornhill (*Trans. First Ind. Med. Cong., Calcutta*, 1895) regarded its ravages in Ceylon as far more serious than those of cholera; this, not on account of the number of deaths it causes directly, but on account of the vast numbers affected, the chronic nature of the disease, and the aggregate mortality, direct, and especially indirect, for which it is responsible.

The parasite (Fig. 91).—The normal habitat of *A. duodenale* is the small intestine of man, particularly the jejunum, less so the duodenum, rarely the ileum or lower reaches of the alimentary canal; very occasionally it is found in the stomach. In these situations it attaches itself by means of its powerful buccal armature to the mucous membrane, from the blood of which it obtains a plentiful supply of nourishment. It is supposed to shift its hold from time to time, the abandoned bite continuing to





ooze blood for a short period. It is said to be very prodigal of the blood it imbibes, the red corpuscles passing through its alimentary canal unchanged, the plasma alone being utilised.

The male and female ankylostomes—present generally in the proportion of one of the former to three of the latter—do not differ so much in size as is the case with many of the other nematodes. The male (Fig. 92) measures from 6 to 11 mm. in length by .4 to .5 mm. in breadth; the female 7 to 15 mm. in length by 1 mm. in breadth. Both sexes are cylindrical in form, white when they are alive, grey when dead, reddish brown when full of blood. In both sexes the posterior end is the broadest part,

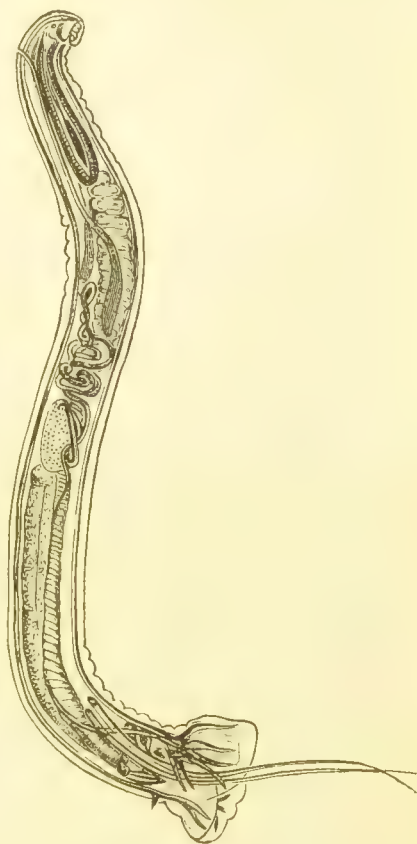


Fig. 92.—Male *Ankylostomum duodenale*.

whence the body tapers forwards to a narrow neck ending in a powerfully armed, bulging and distinct mouth capsule. The margin of this remarkable organ is furnished with four strong, claw-like hooks—two on each side of the ventral line, and two conical teeth—one on each side of the dorsal line. The tail of the female is conical, and ends in a short delicate

spine; the anus being subterminal, and the vagina opening on the ventral surface at the commencement of the posterior third of the body. The tail of the male is provided with a large umbrella-like, trilobate bursa possessing eleven ribs. Two long and very delicate spicules project from the cloaca which opens at the bottom of the bursa. Owing to the relative positions of the sexual openings, the worms in conjugation look like the Greek γ .

Reproduction and mode of infection.—The female ankylostomes produce a prodigious and never-ending stream of eggs (Fig. 90, c) which pass out in the faeces. As already stated, while in the body of the host the development of the embryo does not advance very far; but on leaving the human host it proceeds, in suitable circumstances, so rapidly that in one to two days a rhabditiform embryo (.2 mm. by .014 mm.) is born. This minute organism is very active, voraciously devouring what organic matter it can find and, for a week, growing rapidly (to .56 mm. by .024 mm.). During this time it moults twice. After the second moulting it passes into a torpid condition, in which it ceases to eat, and growth is suspended. In this state it may live for weeks or months, moving about more or less languidly in muddy water, in mud, or in damp earth. Should chance so determine, it is finally transferred to the human alimentary canal, either in muddy drinking water, or in the mud or dirt adhering to the hands or food dishes of the agriculturist, the brick-maker, or other operative engaged in handling the soil; or, it may be, in earth deliberately eaten by the geophagist and children. Arrived in its final host, after moulting again at the end of five weeks (Leichtenstern) it acquires sexual characters and the permanent adult form. The duration of the life of *A. duodenale* in the intestine has not been determined; some state it in months, others in years (Sonsino)—one to three. On account of liability to reinfection, this point, an important one as affecting prognosis, is difficult to determine.







Giles holds that *A. duodenale* may become sexually mature, assuming rhabditiform characters while outside the human body and in the free state; in other words, that it is heterogenetic, an abundant supply of food favouring non-parasitic multiplication. His observations, so far as we are aware, have not been confirmed; though partially upheld by Sandwith, doubt has been thrown on their relevancy by Sonsino and Macdonald.

ANKYLOSTOMIASIS.

History.—Although Griesinger had previously shown that Egyptian chlorosis was due to the presence of the ankylostomum in the small intestine, and although similar observations had been made on negroes in America, it was not until the very fatal epidemic of anæmia among the miners in the Saint Gothard tunnel (in 1880) had called the attention of European observers to the subject, that the real pathological importance of this parasite began to be properly apprehended. Multiplied observations have now shown that, although very minute, this blood-sucking parasite, if present in large numbers and for a length of time, more especially if its victims are poorly fed, is a very dangerous one indeed. The constant drain of blood which its presence entails, the catarrh arising from the irritation caused by the wounds it inflicts on the mucous membrane, the consequent impairment of nutrition, and, possibly, as has been suggested, the absorption of some hæmolytic toxin—the product of the parasite—give rise to a grave cachexia, disqualifying to a greater or lesser extent the subject of it for work, and, not infrequently, leading to a fatal issue.

Its importance.—It is not in every instance in which the ankylostomum is present that consequences so serious ensue. There may be dozens of ankylostomes in the intestine without any appreciable anæmia, or, indeed, symptoms of any description whatever. Grave symptoms are the exception. One

must be careful, therefore, to avoid concluding that the ankylostomum is the cause of every pathological condition with which it may chance to concur.

On the other hand, many inhabitants of tropical and subtropical countries are in a state of chronic starvation. Living on coarse, bulky, innutritious food, they are prone to dilatation of the stomach and dyspeptic troubles. In such, any additional cause of malnutrition, as a swarm of ankylostomes, and a daily though perhaps small loss of blood, may be sufficient to turn the scale against them. In those countries, as elsewhere, there are many who live just on the borderland between health and disease; to such the ankylostomum may prove the last straw that breaks the camel's back.

It is evident that as a complication in typhoid, in kidney disease, in dysentery, in malaria, in fact in any chronic or exhausting disease, the importance of this anæmia-producing parasite cannot be ignored.

The practitioner in the tropics, therefore, must be constantly on the outlook in all cases of anæmia, of dyspepsia, and of debilitated conditions generally, for the ankylostomum. He must bear in mind that this parasite, as will be presently pointed out, if permitted to remain in the intestine for a length of time, may be the cause, not only of remediable anæmia, but of anæmia-produced degenerations of various organs from which recovery is impossible. On this account, also, its early recognition becomes a matter of the first importance.

Further, ankylostomiasis is an important disease from the standpoint of the employer of native labour. The invaliding and inefficiency which it causes among coolies, not to mention the deaths, are often financially a serious matter to the planter and the mine-owner. To them, any wisely directed expense or trouble undertaken for the treating and controlling of this helminthiasis will be abundantly repaid by the increased efficiency of the labourer.

Symptoms.—The essential symptoms of ankylo-



stomiasis are those of a progressive anæmia ; an anæmia which is generally associated with dyspeptic trouble, but which, in uncomplicated cases, is not associated with wasting. If the progress of a case be unchecked, serous effusions in different organs and fatty degeneration of the heart ensue, and death may occur from syncope or from intercurrent complication.

One of the earliest symptoms of an extensive ankylostomum invasion is pain or uneasiness in the epigastrium. This is generally increased by pressure, but, for the time, may be relieved by food. The appetite, sometimes defective, is more often ravenous ; but its gratification is apt to give rise to dyspeptic trouble of various kinds, to colic, to borborygmus, and perhaps to diarrhœa of imperfectly-digested food. Constipation may be present in some instances, irregularity of the bowels in others. The taste may be perverted, some patients exhibiting and persistently gratifying an unnatural craving for such things as earth, mud, lime—what is called pica or geophagy. The stools sometimes, though rarely, have a reddish brown tinge from admixture of half-digested blood. Sometimes they may contain small flakes of blood-tinged mucus. Pure blood is rarely passed. An extensive hæmorrhage, unless there be concurrent colitis, is still more rare, although *post mortem* considerable quantities of blood may be found in the small intestine. Fever of an irregular, intermitting, or even of a sub-continued type, is common. On the other hand, the temperature may be constantly sub-normal. Or these conditions may alternate. After a longer or shorter time, symptoms of profound anæmia gradually disclose themselves. The mucous surfaces and the skin become pallid, the face is puffy, and the feet and ankles are swollen. All the subjective symptoms of a definite anæmia now become more and more apparent ; there is lassitude, breathlessness, palpitations, tinnitus, vertigo, dimness of sight, mental apathy and depression, liability to syncope, and so forth. The circulation is irritable and hæmic bruits

can be heard over the heart and larger blood-vessels. Ophthalmoscopic examination may reveal retinal hæmorrhages.

From some of these symptoms, were it not that with the advancing anæmia there is no loss of weight, one might be led to suspect the possibility of tuberculous or cancerous disease or of Bright's disease. So far from losing weight, the patient may appear quite plump and, though hæmocytometric estimates testify to a slow and steady fall in the corpuscular richness of the blood until the lowest limit compatible with life is reached, there is no true poikilocytosis as in idiopathic pernicious anæmia, no excessive leucocytosis as in leucocythæmia, and not necessarily any enlargement of lymphatic glands, liver, or spleen. The hæmoglobin value of the corpuscles is not depressed correspondingly to the fall in their numbers.

The rate of progress is very different in different cases. In a few a high degree of anæmia may be attained, and even a fatal issue ensue, within a few weeks or months of the appearance of the first symptoms. Such rapid cases are rare; more frequently the disease is an exceedingly chronic one, ebbing, or flowing, or slowly progressing through a long series of years.

Should serious ankylostomiasis occur before puberty, the growth and development are apt to be delayed and stunted.

Diagnosis.—Provided its presence be suspected, ankylostomiasis is easily diagnosed. In tropical countries, anæmia without apparent cause should always suggest a microscopical examination of the fæces. If the ova (Fig. 90, c) of *Ankylostomum duodenale* are discovered, and no other reason for the anæmia be made out, the presumption is that the parasite is at the root of the mischief; at all events, no harm is likely to result from treatment based on this supposition. On the other hand, if no ova are found it must not be concluded that the case is not one of ankylostomiasis; for it sometimes happens that, in

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the later stages of the disease, symptoms will persist although the parasites which caused them in the first instance have disappeared spontaneously, or have been got rid of by treatment. Permanent degeneration of the alimentary canal, of the heart, liver, kidneys, and blood-forming organs may remain, and even prove fatal, although the primary cause is no longer present. The history of the absence of other sources of anæmia is all we may have to go upon in such circumstances.

Ankylostomiasis is sometimes confounded with beriberi, and *vice versa*. The presence of paretic and cardiac symptoms not accounted for by the moderate degree of anæmia in beriberi, and their absence in ankylostomiasis, suffice for diagnosis. The anæmia of ankylostomiasis differs from that of malaria, inasmuch as the latter is accompanied by enlargement of the spleen, a sallow and perhaps pigmented complexion, an icteric tint of the scleræ, occasional attacks of well-marked periodic fever controlled by quinine, and, especially, the presence at such times of the plasmodium in the blood. Of course, ankylostomiasis and malarial cachexia may concur, and often do concur, in the same individual.

Some idea of the intensity of the affection may be got from an enumeration of the eggs in a given quantity of fæces; according to Grassi and Parona, 150 to 180 eggs per cubic centigramme indicate an infection of about a thousand worms, male and female.

Pathological anatomy and pathology.—

As already mentioned, the bodies of the victims of ankylostomiasis are not wasted; on the contrary, there is plenty of fat in the usual situations. The appearance of plumpness is further increased by a greater or lesser amount of general œdema. There may be effusions in one or more of the serous cavities. All the organs are anæmic. The heart is dilated and flabby, its muscular tissue being in a state of pronounced fatty degeneration. The liver, also, is fatty and so are the kidneys.

If the *post-mortem* examination be made within an hour or two of death, the ankylostomes, in numbers ranging from a few dozens up to many hundreds, will be found still attached by their mouths to the mucous surfaces of the lower part of the duodenum, the jejunum and, perhaps, of the upper part of the ileum; but if the examination has been delayed for some hours, the parasites will have dropped their hold, and are then to be found lying in the mucus coating the inner surface of the bowel. Many minute extravasations of blood—some fresh, others of long standing—are seen in the mucous membrane, a minute wound in the centre of each extravasation representing the point at which the parasite had been attached. Sometimes blood-filled cavities, as large as filberts, are found in the mucosa; each cavity enclosing one or two worms and, probably, communicating by means of a small hole with the interior of the intestine. Old extravasations are indicated by punctiform pigmentations. There may be evidence, in the shape of vesiculations and thickening of the mucosa, of a greater or lesser degree of catarrh. Occasionally, streaks or large clots of blood are found in the lumen of the bowel.

Daniels (*British Guiana Medical Annual*, 7th issue, 1895) and others report that microscopic examination of the liver and kidneys shows the presence, within the cells of the parenchyma, of grains of yellow pigment having the reactions of hæmatoidin; indicating an intravascular blood destruction, such as occurs in pernicious anæmia and other diseases in which excessive hæmolysis is a feature. On this account, and also because he finds granules of a ferrous nature in the liver cells, Daniels concludes that the anæmia in ankylostomiasis is, in a measure, the result of blood destruction within the vessels by some toxic substance produced by the parasite and absorbed from the bowel. These results have not been confirmed by other observers; on the contrary, the late Dr. Beaven Rake (*Journal of Pathology*, Nov., 1894)

concluded, from careful estimates of the amount of iron in the livers of five cases of ankylostomiasis, that in this disease the hepatic iron is below the normal average, and that the anæmia is entirely owing to the direct abstraction of blood by the parasites. The occurrence of the condition known as bothriocephalus anæmia lends a certain amount of probability to the toxin-hæmolysis theory. Further observations are necessary before this question can be decided.

Treatment.—*Male fern.*—Until the introduction of thymol by Bozzolo in 1880, extract of male fern was the anthelmintic usually employed in ankylostomiasis. Male fern has still its advocates. It may be used where thymol fails—an occasional occurrence.

Thymol.—Before the administration of thymol the patient should be put on liquid diet for a day or two, and have the bowels well cleared out by an aperient. In the morning, and following the action of the aperient, three or four ten- to thirty-grain doses of well triturated thymol, in cachets, in capsules, or in emulsion, are given on an empty stomach at intervals of an hour. If the bowels do not open spontaneously within four or five hours of the last dose another aperient dose should be given. Usually, by this treatment many ankylostomes are expelled and may be found in the motions. One such course of thymol may suffice; but it is well, after a week has elapsed, again to examine the stools microscopically, and, if it be found that ova are still being passed, to repeat the course of thymol once or oftener.

Certain precautions have to be observed in employing this drug. At times it gives rise to a very unpleasant form of intoxication—vertigo, excitement, etc., and the urine may become dark, as in carbolic acid poisoning. It is advisable therefore for the patient, while taking the drug, to keep his bed and to lie down for several hours after the last dose. Thymol is very insoluble in water and is therefore, in ordinary circumstances, not readily absorbed in poisonous quantities; should, however, the patient, while thymol is

present in the stomach, partake of any alcoholic drink, there is considerable risk of poisoning ensuing. Alcohol, ether, glycerine, turpentine, chloroform and oils are all solvents of thymol, and must therefore be avoided when this drug is being exhibited. Thornhill related an instance in which a fatal result was brought about, apparently, by neglect of the obvious precautions suggested by these facts. A man had received thirty grains of thymol in water at 7 a.m. "He experienced no special symptoms after it, and at 9 a.m. the nurse gave him a second dose of thirty grains. As this man was supplied with arrack as an extra, and as in such cases a portion of the arrack was usually given at 9 a.m., the nurse gave it to him just after administering the second dose of thymol. The result was that intense collapse set in almost at once, and, notwithstanding all efforts, the man died within twenty-four hours, the collapse manifestly being due to the arrack dissolving the thymol which was thus absorbed." Thornhill mentions two additional fatal cases of thymol poisoning occurring in his experience; other writers have recorded similar fatalities. For this reason, and because it is an extremely unpleasant drug to take—sometimes giving rise to severe burning in the stomach, throat and gullet, and, not infrequently, to excitement, giddiness, fainting and vomiting—an equally efficient but safer drug is a desideratum.

Without careful preparation by rest and judicious feeding, thymol must on no account be used in advanced cases of ankylostomiasis and where prostration is extreme.

Other drugs.—Thornhill seemed to prefer male fern to thymol as being somewhat less dangerous, less disagreeable to swallow, and, possibly, equally if not more efficacious. When male fern is given, the preparation of the patient, the dose, and the mode of administration are the same as for thymol. Oil of peppermint, kerosene in thirty-drop doses, and carbonate of guaiacol have all been suggested as





substitutes for thymol and male fern ; as yet we have no report of any extensive trials having been made of these drugs.

Powell (*Ind. Med. Gaz.*, 1899) considers that betel-nut chewing acts as a prophylactic to ankylostomiasis, and recommends supplementing the thymol treatment by ounce doses of powdered areca nut.

Convalescence.—The dieting of convalescents from ankylostomum disease must, for a time, be very carefully conducted. A rich, full dietary is to be avoided until the powers of digestion have in a measure become re-established ; otherwise, enteritis and diarrhœa may prove very troublesome and retard recovery—perhaps prevent it altogether. Iron and arsenic are indicated as blood restorers.

Prophylaxis.—In devising a system of prophylaxis for ankylostomiasis, the fact that it is by means of the fæces of the already infected that the parasite is spread must be kept prominently in view. Fæcal contamination of the soil and water must therefore be prevented. The promiscuous depositing of fæces about huts, villages, and fields must be interdicted. Abundant and easily accessible privy accommodation must be provided in coolie lines, in miners' camps, in native villages, and along the highways of traffic. In the absence of a more elaborate system of conservancy, pits or trenches will suffice. They may be filled up with earth and fresh ones opened from time to time. I believe the Chinese plan of storing night-soil in large, cemented, water-tight pits is a good one. It is known that if the ova of the ankylostomum are kept in pure fæces the embryo is developed and escapes from the egg in due course ; but it is also known that, unless the embryo be supplied with a certain amount of air and earth, it soon dies. The thing to be avoided, therefore, is the mixing of *fresh* fæces with earth. In the Chinese system, night-soil is first stored in the pits for months on end ; in these it ferments and rots before being finally spread on the fields. In this way the embryos

of the ankylostomum are killed and, at the same time, a valuable fertiliser is secured for the agriculturist.

It is manifest that in devising privies and sanitary regulations, the habits of the people they are intended to benefit must be taken into account ; if this be not attended to, if native habits and prejudices are ignored, any system, no matter how perfect it may be in theory, will fail in practice.

The water supply should also be carefully guarded from all possible sources of faecal contamination. Drinking water should be boiled or strained. So far as possible, facilities for removing all earth and mud from the hands and dishes before food is partaken of, should also be provided and their use encouraged.

Badly contaminated ground had better be abandoned. If this should be found impracticable, the soil had better be turned over with the plough, or roasted with grass fires, or treated in such a manner that any ova or embryos it may contain are destroyed or buried. The systematic periodical inspection of plantation coolies is to be recommended. At these inspections, all subjects of anæmia or dyspepsia should be put aside for more careful examination ; if the ova of ankylostomes are found in their faeces a judicious dosing with thymol may avert serious disease in the individual, and also prevent him from becoming a source of danger to his companions.

In view of the great danger to health that exists in certain countries from this and similar parasites, the sanitary authorities in such places ought to circulate among the people, by means of printed leaflets or posters, a few simple directions for the prevention of ankylostomiasis and kindred diseases.

RHABDONEMA INTESTINALE.

It sometimes happens that, while searching the faeces for the ova of the ankylostomum, the observer is astonished by seeing a small, snake-like animal (Fig.







93, *a*) suddenly rush across the field of the microscope. On careful examination, this animal is found to be about 0·2 mm. or 0·3 mm. in length by 0·013 mm. in breadth; to have a sharply pointed tail and a

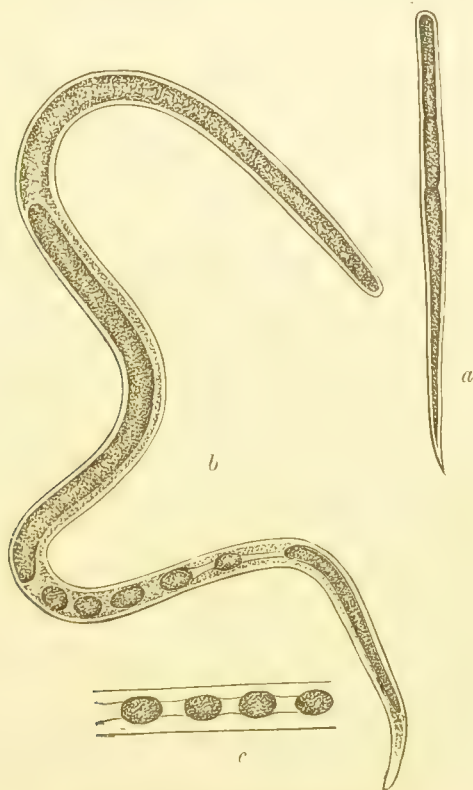


Fig. 93.—*Rhabdonema intestinale*.

rounded head; to be transparent; and to exhibit a short œsophagus which terminates in a double œsophageal bulb, the posterior end of which is provided with three tooth-like segments. This is, or rather was, called *Anguillula stercoralis*.

Anguillula stercoralis was discovered by Normand in 1876. For a time it was supposed to be a cause of a form of chronic diarrhœa very prevalent in Cochin China. Later investigations, while clearing up the

natural history of the parasite, have robbed it of any claim to pathological importance. It has been found that, though the anguillula is not so common, its geographical distribution is about coextensive with that of *Ankylostomum duodenale*, and that the physical conditions demanded for the non-parasitic stages of these two worms are about the same. Powell found it in India in about fifteen out of every twenty cases of anæmia. It has been established that this so-called *Anguillula stercoralis* is the rhabditiform phase of *Rhabdonema intestinale* (Fig. 93, b) (formerly called *Anguillula intestinalis*), a nematode which, in its parasitic form, lives in the mucus of the duodenum and upper part of the jejunum of man.

The mature rhabdonema is a minute, slender worm, measuring 2 mm. in length by .06 mm. in breadth. No male parasite has been discovered. The adult female is readily recognised by her dimensions, and by the string of five or six ellipsoidal eggs (.01 mm. by .034 mm.) visible about the centre of the body.

On being passed into the contents of the bowel the ova develop with great rapidity ; so that before leaving the host, unless during violent purgation, the embryo has escaped and is swimming about in the fæces, as already described, with great vigour, especially when these are fluid. Only in the event of violent purging do the ova appear in the stools ; they may in this way be hurried out of the host before the embryo has escaped. Such ova are readily recognised by the way in which they are strung together, end on end, inside a delicate tube (Fig. 93, c). On leaving the host, unless they have access to some non-putrefying fluid, the embryos soon die ; it is necessary, therefore, if we would follow their further development, to mix the fæces with water. If this mixture be kept at a low temperature the young rhabdonema develops into a filariform larva which, on being swallowed by man in drinking water, quickly assumes the mature parasitic form already alluded to. If, on the contrary, the cultivating medium be kept









at a higher temperature, the embryos develop into male (·7 mm.) and female (1·0 mm.) rhabditic forms which, in time, produce in their turn filariform larvæ, similar to those obtained directly from the embryo in cold climates and capable, on being swallowed by man, of developing into mature *Rhabdonema intestinale*.

The rhabdonema may produce some slight degree of catarrhal irritation of the bowel, though even this is doubtful. They have been found occasionally coiled up in the intestinal follicles, but no serious lesion can with certainty be attributed to their presence; a fortunate circumstance, as hitherto the use of anthelmintics has not proved effectual in procuring their expulsion. Sonsino recommends the prolonged exhibition of liquor ferri perchloridi in combination with small doses of thymol. It is probable that catarrhal conditions of the bowel favour these parasites, and that this is the reason for their being so frequently encountered in cases of chronic tropical intestinal flux. Treatment should therefore be directed to the cure of the catarrh.

The prophylaxis for rhabdonema is the same as that recommended for *Ankylostomum duodenale*.

STRONGYLUS SUBTILIS.

Looss (*Centralblatt für Bakteriologie*, xviii., p. 161, 1895) has described a very delicate nematode frequently encountered in Egyptian fellahs. Its habitat is the upper part of the small intestine. The male, which is provided with two spicules, is from 4 to 5 mm. in length by 0·07 mm. in breadth; the female is slightly larger and is much more abundant than the male. The eggs are oval, thin-shelled, with an unsegmented vitellus, and measure $63\ \mu$ by $41\ \mu$. This parasite does not occur in large numbers; and, as its mouth is unarmed and its dimensions are exceedingly minute, it does not appear calculated to give rise to any particular symptoms.

TREMATODES.

AMPHISTOMUM HOMINIS (Fig. 94).

This minute trematode, discovered in the cæcum and colon of two Indians in Calcutta by Lewis and MacConnell in 1876, measures 5 to 8 mm. in length by 3 to 4 mm. in breadth. It is readily recognised by its large posterior sucker, which is terminal and greatly exceeds in breadth the rest of the body. It attaches itself to the mucous membrane of the bowel by means of this sucker. The ova, which are operculated at the smaller end, average $150\ \mu$ in length by $72\ \mu$ in breadth. Nothing is known of the life-history.



Fig. 94.—*Amphistomum hominis*.
(Nat. size.)

This trematode has been found by Giles in natives of Assam, and by Law in an East Indian immigrant in British Guiana.

DISTOMUM BUSKI (V. CRASSUM) (Fig. 95).

Distomum buski, over an inch in length, is the largest of the distomes inhabiting man. It has been found in China, Sumatra, the Straits Settlements, Assam, India, and by Law in an East Indian immigrant in British Guiana. It is an elongated oval in shape, rather narrower anteriorly than posteriorly, fleshy, and covered with minute spines. The suckers are placed close together. The ova are oval, $125\ \mu$ by $75\ \mu$, and are closed, I find, by a very delicate operculum. In two recorded instances (Cobbold) *D. buski* was associated with attacks of recurring diarrhoea and other signs of intestinal irritation. It probably inhabits the upper part of the small intestine. Nothing is known of



Fig. 95.—*Distomum buski*. (Nat. size.)

the life-history. It may be expelled by thymol given as for ankylostomum.

DISTOMUM HETEROPHYES (Fig. 96).

Distomum heterophyes is the smallest distome, so far as known, inhabiting man. It measures 1 to 1½ mm. in length by 0·7 mm. in breadth. It is of a bright red colour. The anterior, narrower, and more mobile part of its body is covered with minute spines. The eggs are reddish brown, and measure 26 μ by 15 μ . It was discovered by Bilharz in 1851, in Cairo, in the small intestine of a child, the little parasites looking like red points on the mucous surface. Since that time it has been found in Egyptians by other observers. Nothing is known of its life-history. It is believed to be innocuous.

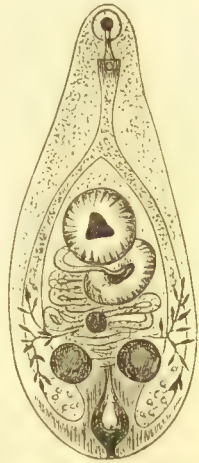


Fig. 96.—*Distomum heterophyes*. (Magnified.)

CESTODES.

The ordinary tape-worms, *Tenia saginata* and *Tenia solium*, and their respective cystic forms, are common enough in the tropics and sub-tropics, their distribution being regulated by the presence or absence of their proper intermediary hosts—the ox in the one case, the pig in the other—and by the habits of the people as regards cooking and conservancy. *Tenia echinococcus* of the dog, and its cystic form—hydatids—are found wherever the dog and sheep are found, that is, practically, everywhere. *Bothriocephalus latus* is known to occur in Turk-estan, in Japan—where the natives are in the habit of eating raw fish—and among the natives on the shores of Lake 'Ngami, South Africa. Ichthyophagous habits are probably responsible for the occurrence of the large tape-worm described by Blanchard

under the name *Krabbea grandis*, which Ijima and Kurimoto found in a Japanese from the province of Hizen (*Comptes rendus de la Soc. de Biologie*, I., p. 609, I., 94).

The only cestodes of man which, so far as known, have any claim to be regarded as more or less special to warm climates are *Tenia nana*, *Tenia madagascariensis*, and *Bothriocephalus mansoni*. Doubtless there are other species which, so far, have escaped observation.



TENIA NANA (Fig. 97).

This minutest of the tape-worms inhabiting man was first found as a human parasite by Bilharz, in Cairo, in 1852. In recent years it has been identified with *Tenia murina* of the rat and other rodents. It has a somewhat extensive geographical distribution. In the case of man, at all events, it seems to have a predilection for warm countries. Besides Egypt, it has been found in Italy, Buenos Ayres, and in Bangkok, Siam. It is readily recognised by its small size—10 to 15 mm. in length with a maximum breadth of 00·7 mm. It has from 150 to 200 joints, the head being furnished with a retractile proboscis surrounded by a single row of from twenty-four to twenty-eight hooklets. The ova are oval or round, $30\ \mu$ to $37\ \mu$ in diameter; they are possessed of three membranes, within which the hexacanth embryo can with difficulty be made out.

Fig. 97.—*Tenia nana*. (Magnified.)

Grassi has shown that in the rat the embryo of this cestode can dispense with an intermediary host, and that it penetrates the mucous membrane of the intestine, and there develops into its cysticercus.

Jonia Africana n. sp. *Lauro* p. 229, 1901
Contr. Bot. f. Mat. L. Pac. l. abg. Bd. xxviii 1900, p. 48

After a time this cysticercus ruptures, permitting the head to escape and attach itself to the mucous membrane, where it speedily grows into the mature tape-worm.

Cases are on record in which the human intestine was beset with hundreds of these minute parasites which, by their presence, gave rise to marked symptoms of gastro-intestinal irritation. They are readily expelled by male fern.

TENIA (DAVAINEA) MADAGASCARIENSIS.

Discovered by Grenet in Mayotte (Madagascar), this parasite has since been found in Mauritius, in Bangkok, and by Dr. Daniels (*Lancet*, vol. ii., p. 1455, 1896) in a Demerara Indian. On account of its wide geographical range Blanchard (*Bul. de l'Acad. de Méd.*, Jan. 12, 1897; *Archives de Parasitologie*, April, 1899) shrewdly remarks that its intermediary host is probably some insect of similar distribution; possibly, he suggests, one intimately associated with ships—the cockroach (*Periplaneta orientalis*), for example.

Tenia madagascariensis attains a length of from 25 to 30 cm., and is made up of from 500 to 600 trapezoid joints. The head terminates in a massive rostellum surrounded by a double circle of ninety hooklets 18 μ in length. The suckers are round and of considerable size. The genital pore is unilateral. In the ripe segments the ova are agglomerated into a number of separate rounded masses.

So far as is known, the symptoms produced by this parasite correspond with those caused by the more familiar tape-worms. It is probably amenable to flinmas.

BOTHRIOCEPHALUS MANSONI (Fig. 98).

This parasite, the larval form of an unknown species of bothriocephalus, was discovered by the writer in making the *post-mortem* examination of a

Chinese in Amoy. A number of specimens were found lying under the peritoneum in the neighbourhood of the kidneys and iliac fossæ, and also one, apparently a free specimen, in the pleural cavity. They were more or less coiled up and irregularly disposed in the subperitoneal fascia, looking like ribbon strings of fat until they were turned out, when they exhibited feeble yet distinct movements. No differentiated head, no definite structure, and no evidence of sexual organs could be discovered; neither was there any attempt at segmentation visible. When fresh, the parasites measured about 30 to 35 cm. in length by about 2.5 mm. in breadth. They were flat, and tapered slightly towards one end. At the broader end there was a sort of papilla which, in some instances, was retracted. Scheube extracted a similar parasite from the urethra of a Japanese; it has also been found in Bathurst, Australia. Daniels has quite recently found this or a similar parasite in a Carib in British Guiana.

Fig. 98. — *Bothriocephalus mansoni*. (From specimens preserved in spirits and therefore shrunken.)

Leuckart suggests that the definitive host is probably a carnivorous animal closely associated with man.

LARVÆ OF DIPTEROUS INSECTS.

A residence in the alimentary canal of some vertebrate animal is a regular feature in the life-history of many dipterous insects. The ova of the insect are

either licked from the skin, or swallowed in the food on which they had been deposited. In this way they get transferred to the stomach where, after a time the larva is hatched out and proceeds in development. In due course it appears in the fæces. Man is not infrequently victimised in this way, especially in tropical countries. Sometimes, until a correct diagnosis is arrived at, not a little alarm is caused by the appearance of these creatures in the stools. They are easily recognised. The ringed, cylindrical body, from $\frac{1}{2}$ inch to 1 inch in length according to species, broad at one end, tapering at the other, and usually beset with little spines or hairs, is sufficiently diagnostic (Fig. 99). A dose of castor oil will probably expel any that may not have been passed spontaneously.

A rational prophylaxis would consist in the covering up of food after it has been cooked, in order to prevent the access of flies.

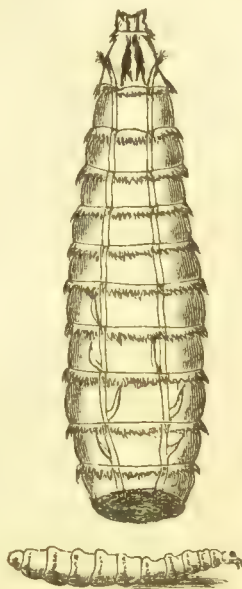


Fig. 99.—Larvæ of *Musca vomitoria*, the blue-bottle fly. (Upper figure magnified.) (Leuckart.)

SECTION VI.—SKIN DISEASES.

CHAPTER XXXIX.

DISEASES OF THE SKIN.

I.—*Non-specific.*

PRICKLY HEAT.

PRICKLY heat or, as it is sometimes called, lichen tropicus, is probably a form of miliaria (not of lichen) connected with the excessive sweating incident to the heat of tropical climates. According to Pollitzer, the mechanism of its production depends on the non-cornification of the cells of the stratum corneum, the individual cells of which swell and so obstruct the orifices of the sweat glands, thereby leading to accumulation of sweat in the ducts. The swollen condition of the cells of the stratum corneum and their non-cornification arise, he maintains, from their being sodden by constant perspiration. Pearse (*Journ. Trop. Med.*, June and October, 1899) regards the disease as an acute distension of the sebaceous glands by their own secretion; the glands, he holds, are over-stimulated in order to supply an adequate amount of sebum to the skin, so as to make up for the loss of that material washed away by inordinate sweating.

Nearly every European in the tropics suffers from prickly heat, particularly during the earlier years of residence. Some never seem to become acclimatised in this respect, but continue year after year to exhibit their crop of prickly heat when the hot season comes round.

Though sufficiently annoying in the robust and healthy, in them prickly heat is not a grave affair. It

Lately Durham (Liverpool School of
Trop. Med. Memoir VII) has advanced the
view that it is an infective disease
depending on a minute amoeboid
~~parasitic~~ bodies which are present in
the contents of the varicels before them
have become clouded by leucocytes.
The disease is acquired by injection &
spreads by auto-infection and is readily
cured by application of weak solutions
of iodine or ^{ammonia} of solutions 1:500 or 1:1000
of carbolic solution. The latter
with or without pyrexia being rubbed
thoroughly in with a soaked pledget of
cotton wool.

is otherwise in the case of the invalid, of delicate sickly children, of hysterical and, especially, of parturient women; to these it may prove, by interfering with sleep and provoking restlessness, a very serious matter. Prickly heat is also a common though indirect cause of boils; for the breaches of surface, following on the scratching it induces, afford many opportunities for the invasion of the micro-organisms of that disease.

Prickly heat consists of a miliary-like eruption, generally most profuse on those parts of the body, as around the waist, which are closely covered with clothing; but it also occurs on the backs of the hands, arms, legs, forehead, occasionally on the face, the scalp, in fact on any part of the surface of the body except the palms and soles. The minute, shining, glass-like vesicles, and the numerous, closely set, and slightly inflamed papules give the skin the feeling as if thickly sprinkled with grains of sand. The eruption may keep out for months on end, becoming better or worse according to circumstances. The pricking and itching are often exceedingly distressing. Anything leading to perspiration immediately provokes an outburst of this almost intolerable itching—nothing more certainly than a cup of hot tea or a plate of hot soup. Long drinks, exposure to the hot sun, close rooms, warm clothing, all aggravate the distress. Sometimes the little vesicles may pustulate, doubtless from micrococcus infection. So soon as the weather becomes cool the eruption and the irritation quickly subside.

Treatment.—Manifestly the most important thing is the avoidance of all causes of perspiration—particularly the copious consumption of fluids especially hot fluids, moderation in exercise, avoiding close rooms, warm clothing, and so forth. Soap should not be used in the bath. The sleeping mattress and pillow should be covered with a finely woven grass mat, and the bed provided with what is known in the East as a “Dutch-wife”—that is, a hollow cylinder, 4 feet by 8 or 10 inches, of open rattan

work, over which the arms and legs can be thrown and unnecessary apposition of sweating surfaces so avoided. A punkah at night is a great comfort. Many things have been recommended as preventives; for example, rubbing the body over after the bath with the juice of a lemon, Jeyes' fluid or bran in the bath, etc. Every bath-room in the tropics should be provided with some mildly astringent and antiseptic dusting powder. A very good one consists of equal parts of boric acid, oxide of zinc and starch. This should be freely applied after careful drying of the skin, particularly to the axillæ, crutch, under the mammæ in women, and between the folds of skin in fat children and adults. A simple precaution of this sort saves much suffering both from prickly heat and epiphytic skin disease. Pearse strongly recommends the inunction of a mixture of almond oil and lanoline in the proportion of 8 to 1, and scented according to fancy. St. George Gray finds thin flannel a better wear than cotton or linen as a preventive of prickly heat. Sometimes the following powder, gently rubbed in for five or ten minutes with a damp sponge, cures bad patches of prickly heat: Sublimed sulphur, 80 parts; magnesia, 15 parts; oxide of zinc, 5 parts. Lotions of calamine, with or without hydrocyanic acid, or of carbolic acid, relieve the itching temporarily.

Durham's treatment

II.—Caused by bacteria.

TROPICAL SLOUGHING PHAGEDÆNA.

Definition.—A rapidly spreading, but generally after a time spontaneously arrested, gangrene of the skin and subjacent tissues, resulting in the formation of a large sloughing sore. Though occasionally fatal, these sores, as a rule, under favourable conditions, granulate and cicatrise, or become chronic ulcers.

Geographical distribution. — Sloughing phagedæna is common in most tropical countries, particularly in those with a hot, damp climate.

These sores are often named after those districts in which they are specially prevalent; thus we hear of Mozambique ulcer, Yemen ulcer, etc. They are found principally in jungle lands, less frequently in towns and well-settled districts. Whether tropical sloughing phagedæna, and the hospital gangrene at one time so prevalent in the hospitals of Europe, are the same disease, it is difficult to say. They agree in some respects; but in the marked tendency of the tropical sore to self-limitation, and, possibly, as Scheube points out, in its relatively feebly infective power, there is some indication of a specific difference.

Ætiology.—Doubtless depending on the proliferation in the affected tissues of some specific micro-organism, not yet satisfactorily separated, the germ of sloughing phagedæna finds its special opportunity in the bodies of men who from overwork, underfeeding, exposure, malaria, dysentery, scorbutus, and the like, are physically depressed. Thus it is apt to attack the half-starved, malaria-stricken pioneers in jungle lands, overdriven slave gangs, and soldiers campaigning in the tropics. In such circumstances a slight wound, an abrasion, even an insect bite, or an old chronic ulcer may serve as the starting-point for one of these terrible sores. Where yaws and sloughing phagedæna are co-endemic, the sores of the former may become infected with the virus of the latter, and serious sloughing and cicatricial contractions result. The feet and legs, being most exposed to injury, are the most frequent locations for this form of ulceration; but the arms or any other part of the body may also be attacked.

Symptoms.—If the disease occur in previously sound skin the first indication is the formation of a large bleb with sero-sanguinolent contents. The formation of this may be attended with some pain and constitutional irritation. When, in the course of a few hours, the bulla ruptures, an ash-grey, moist slough is exposed. The sloughing process rapidly extends in all directions until the skin and

subcutaneous fascia, over an area many inches in diameter, are converted into a yellowish, moist, horribly stinking slough. After a few days the centre of the slough begins to liquefy, the sore still continuing to extend at the periphery. In the course of a week or longer the sloughing process may cease and the slough be gradually thrown off. Then it is seen that not only have the skin and superficial fascia been destroyed, but that possibly muscles, tendons, nerves, vessels, and even the periosteum of the bones have shared in the gangrenous process. Fortunately in many instances the deeper structures are spared, the disease being relatively superficial. Sometimes, however, important structures, including joints, bones and large blood-vessels, are destroyed; in such cases, even if life be spared, great deformity may ensue from different forms of ankylosis, or from strangulation of a distal part by a contracting cicatrix.

When the disease attacks a pre-existing wound or sore, the granulating surface of this becomes dry, and rapidly assumes the appearance and characters of a slough.

In sloughing phagedæna the neighbourhood of the sore is somewhat congested and swollen, particularly so if the patient has been obliged to use the limb. Constitutional disturbance may be considerable and of an adynamic type. On the other hand, it occasionally happens that large sores are attended with singularly slight local or general reaction. In bad cases a sapræmic condition is apt to supervene and carry off the patient; or death may occur from bleeding from the opening of a large blood-vessel.

Treatment.—It is of the first importance to endeavour to correct any cachectic state which may be present. Thus good food, fresh vegetables, lime juice, and quinine are almost invariably indicated. Opium in full doses, not merely to assuage pain, but on account of its special action on the phagedænic process, is usually of great service. Locally, an endeavour must be made effectually to destroy the

germ by the application of some powerful and penetrating caustic to the diseased surface. With this view, on the strength of considerable experience, I recommend that the patient be put under chloroform and the slough thoroughly dissolved off by the free application of pure carbolic acid, a piece of lint on a stout stick being used as a mop for the purpose. Thereafter the limb should be elevated and placed under some improvised irrigator from which a weak, warm antiseptic solution should continuously trickle over the now clean surface. If the phagedenic action recur the carbolic acid must be promptly reapplied as often as may be necessary. On healthy granulations springing up the ulcer is to be treated on ordinary principles.

Patients with this disease should be regarded as infective, and, so far as possible, isolated.

BOILS.

The anatomical and clinical features of this painful affection are too familiar to require detailed description. Suffice it to say that a boil is produced by the proliferation of *Torula pyogenica* or other pyogenic micro-organisms in the skin and subcutaneous tissue; that the organism gives rise to local and limited infiltration of the tissues with lymph which subsequently and rapidly necroses, the necrotic core being surrounded by an areola of acute inflammation; that this core is separated by a process of sloughing and so got rid of, the resulting ulcer speedily healing and leaving a depressed scar, which, when occurring about the legs, may become pigmented. Though a self-limiting disease locally, it is nevertheless capable of being inoculated elsewhere in the same individual, both through a breach of surface and, also, by simple contact of the discharges with the skin, the micro-organism apparently entering by a hair follicle. This auto-inoculability of boils is apt to be overlooked.

Conditions of debility, presumably by lowering resistance, predispose to boils; the subjects of diabetes are specially prone to them, the saccharine state of the blood or secretions seeming to be particularly favourable to growth of the specific germ.

At one time or another few Europeans in the tropics escape an attack of boils. In some instances crop after crop succeeds one another, the individual boils being so numerous that the patient is quite unfitted for work by the attendant pain and fever. In certain years so many members of a community are attacked that the disease may be described as being epidemic. These epidemics, occurring when some particular fruit is in season, are very generally, but probably incorrectly, attributed to the use of the particular fruit in question. Mangos, probably erroneously, are frequently held responsible.

Treatment.—Any constitutional irregularity must be treated appropriately. Malaria demands quinine; anæmia and debility, iron and wine; constipation, aperients; diabetes, a suitable diet. I have never seen any good from such vaunted specifics as calcium sulphuret, tar water, or yeast.

Boils ought never, unless in very exceptional circumstances, to be poulticed. Poulticing, although it may relieve the pain of the existing boil, is prone to be followed by more boils in the area sodden by the heat and moisture. Neither should boils be incised or squeezed. The only exception to the rule for not cutting is in the case of boils occurring in the scalp or in the axilla. In the former situation, unless opened early, they are apt, especially in young children, to burrow and cause troublesome abscesses: in the latter situation boils tend to be very indolent and painful, and do not readily spontaneously break through the lax integuments.

In any situation in which the boil is liable to be irritated by pressure or clothing, it is sometimes a good plan to cover the part with a circle of wash-leather spread with soap plaster, and having a small

hole cut in its centre corresponding to the apex of the boil. When a boil opens, the discharge must be kept from soiling the adjoining skin, and the patient must be warned against touching the skin elsewhere with pus-soiled fingers. The parts must be frequently cleansed with 1 in 1,000 corrosive sublimate lotion, powdered with boracic acid and covered with a dry, absorbent antiseptic dressing. A threatening boil may often be aborted by touching the little initial itching or vesiculated papule with some penetrating antiseptic, as iodine tincture, or by painting it with collodion. A very successful method is to drill slowly into the centre of the papule with a pointed pencil of hard wood dipped in pure carbolic acid. The point of the pencil should penetrate at least an eighth of an inch, and should be frequently recharged with the acid during the drilling process; the pain is trifling. In this way, in a severe attack of furunculosis, boil after boil may be aborted and the attack brought to an end.

Change of air is found necessary at times in severe cases.

PEMPHIGUS CONTAGIOSUS.

Definition.—A non-febrile, highly contagious skin disease peculiar to warm countries. It is characterised by the formation of large vesicles or bullae which are unattended by marked inflammation, ulceration, or the formation of crusts or scars.

Geographical distribution.—*Pemphigus contagiosus* is very common in south China during the hot weather; in some years it may be described as being epidemic. It is perennial in the Straits Settlements, and it is known in Madras, in North Queensland (Finlay), Japan (Munro), and America (Corlett). Doubtless, although it has escaped notice by most medical writers, it is common enough elsewhere in the tropics, or wherever heat and moisture combine to bring about a state of skin favouring its development on the infective material being applied. It is

especially common in schools and similar institutions where large numbers of children are thrown much together; they readily pass the disease one to the other. European children are more prone to it than native children; European adults are by no means exempt, but the native adult is rarely affected.

Symptoms. — *Pemphigus contagiosus* closely resembles certain forms of the *impetigo contagiosa* of temperate countries, and is doubtless a variety of this class of skin disease. The individual lesions, as can readily be ascertained by inoculation experiments, begin as minute erythematous specks which rapidly proceed to the formation of vesicles, bullæ, or even large pemphigus-like blebs. The little blister springs abruptly from sound skin; there is no, or very little, areola of congestion. For a short time the hemispherical bleb is beautifully pellucid, tense, and shining. Presently the serous contents become somewhat turbid, and the blister gets flaccid and dull. At this stage, either from scratching or pressure, the blister is generally ruptured. The morbid process does not at once come to an end, but proceeds as an advancing, eccentrically-spreading exfoliation of the epidermis; an exfoliation which may not cease to advance until an area an inch or more in diameter is denuded of epithelium. Then, in that particular spot, the disease stops, a pinkish, slightly glazed-looking patch, rarely covered with a tissue paper-like scale, remaining for some time. Occasionally, after the rupture of the primary bleb, vesication may continue in the peripheral portion of its remains. Only one or two blebs may be visible on the entire surface of the body; generally there are many, the disease being spread by the fingers in scratching or rubbing.

Pemphigus contagiosus may occur in almost any part of the body. In young children it is usually diffuse; in adults it is mostly confined to the axilla and crutch. In these latter situations it gives rise to much irritation and discomfort, owing to the suc-

cessive crops of bullæ running into each other and rendering the parts raw and tender, and predisposed to boils or some form of eczematous intertrigo. During warm, moist weather it may be kept up indefinitely by auto-inoculation.

Assistant-Surgeon Soorjee Narain Singh describes (*Trans. First Ind. Med. Cong.*, 1895) a series of cases of a form of contagious pemphigus occurring in rapid succession in the children of three families in India (exact locality not specified), which bears some resemblance to the pemphigus contagiosus above described. It differs, however, inasmuch as in the Indian disease the bullæ were very large—often larger than hen's eggs, and persisted for from one to three weeks. In one of the thirteen cases described there followed a certain amount of sloughing at the seat of the bullæ; in the others there was neither ulceration nor constitutional disturbance.

Ætiology and pathology.—Like ordinary impetigo contagiosa, this is undoubtedly a germ disease. I have found a diplococcus in the epidermis and fluid of the blister; whether this is the special bacterium responsible for the disease, cultivation and inoculation experiment have not yet decided.

Diagnosis.—Absence of constitutional symptoms, or a history of such, distinguishes pemphigus contagiosus from chicken-pox. Absence of trichophyton elements and of a well-defined, slightly raised, festooned, and itching margin, together with the presence of large blebs and scaling of the epidermis, distinguish it from ordinary forms of body ringworm; a disease with which, when occurring in the armpits and crutch in adults, it is so frequently confounded.

Treatment.—Cleanliness, the frequent use of a bichloride of mercury lotion (1 to 1,000), and a dusting powder of equal parts of boracic acid, starch, and zinc oxide are speedily effective. In the school and nursery those responsible for the care of children must be informed of the contagiousness of

this unpleasant affection, and measures be instituted accordingly.

III.—*Caused by vegetable parasites.*

MYCETOMA, OR THE FUNGUS FOOT OF INDIA.

Definition.—A disease of warm climates, particularly of India, affecting principally the foot, occasionally the hand, rarely other parts of the body, never the internal organs. It is characterised by enlargement and deformity of the part; an oily degeneration and general fusion of the affected tissues; the formation of cyst-like cavities communicating by sinuses, and containing peculiar mycotic aggregations in an oily purulent fluid which escapes from fistulous openings on the surface. The disease runs a slow course, is never recovered from spontaneously, and, unless removed, terminates after many years in death from exhaustion.

History and geographical distribution.—

The earliest notice of this disease we owe to Kämpfer (1712). Its more modern history commenced with Godfrey, of Madras, who, in the *Lancet* of June 10th, 1843, gave a description of several unquestionable examples under the title, "Tubercular Disease of the Foot." Subsequently Balingall (1855), who was the first to suggest its parasitic nature, Eyre (1860), and others added considerably to our knowledge of the subject. The merit of bringing the disease prominently before the profession, and of distinctly describing its clinical and anatomical features, as well as of suggesting its probable pathology, belongs entirely to Vandyke Carter, who, from 1860 to 1874, in a series of important papers, furnished the information on which all later descriptions have been principally founded. Carter was the first to point out the presence of mycotic elements in the discharges coming from the implicated structures, and in the contents of the characteristic cysts and sinuses with which they are honeycombed. Since the date of Carter's



Fig. 100.—MYCETOMA OF ABOUT TWO YEARS' STANDING. (After Legrain.)

papers, beyond the discovery that the associated fungus, in at least one variety of mycetoma, possesses affinities to actinomyces, as suggested by him in 1886, and that the disease is not confined to India, there has been no very important addition to our knowledge.

In India mycetoma is endemic in districts more or less limited. These districts are scattered over a wide area, the intervening regions—in some instances whole provinces, as that of Lower Bengal—enjoying an almost complete immunity. It appears to be acquired only in rural districts, the inhabitants of the towns being exempt. Among the most afflicted districts may be mentioned Madura—hence the name “Madura foot” by which mycetoma is often known—Hirsar, Ajmeer, Delhi, various places in the Punjab, Kashmir, and Rajputana. In recent years we have accounts of its occurrence with some degree of frequency in Senegambia, and, also, of a very few examples in Algeria, the Soudan, Cochin China, Italy, the United States and in South America. It is probable that in time mycetoma will be found to be endemic in many warm countries in which it has hitherto escaped recognition.

Symptoms.—Mycetoma begins usually, though by no means invariably, on the sole of the foot—most often the right. The first indication of disease is the slow formation of a small, firm, rounded, somewhat hemispherical, slightly discoloured, painless swelling perhaps about half an inch in diameter (Fig. 100). After a month or more this swelling may soften and rupture, discharging a peculiar viscid, syrupy, oily, slightly purulent, sometimes blood-streaked fluid containing in suspension certain minute, rounded, greyish or yellowish particles, often compared to grains of fish roe. In other examples of the disease the particles in the discharge are black, having the size and appearance of grains of coarse gunpowder. Sometimes these particles are aggregated into larger masses up to the size of a pea. In time additional swellings, some of which break down and form similar sinuses,

appear in the neighbourhood of the first or elsewhere about the foot. For the most part the sinuses are permanent, healing up in a very few instances only. Gradually the bulk of the foot increases to perhaps two or three times the normal volume (Fig. 101). There is comparatively little lengthening of the foot ; but there



Fig. 101.—Section of a “Madura foot.” (T. R. Lewis.)

is a general increase in thickness, so that in time the mass comes to assume an ovoid form, the sole of the member becoming convex, the sides rounded, and the anatomical points obliterated. The toes may be forced apart, bent upwards at the tarso-phalangeal joints or otherwise misdirected ; so that, on the foot being placed on the ground, the toes do not touch it. The surface of the skin is roughened by a number of larger or smaller, firmer or softer, hemispherical elevations, in some of which the orifices of the numerous sinuses open. Most of these orifices are easily made out : others are not so apparent, their position being indicated and, at the same time, concealed by a bunch of pale, flabby, fungating, and but slightly vascular granulations. In the latter the orifice may

be hard to find. Once the probe is got to enter, the instrument readily passes to a considerable depth, even to the bone ; in advanced cases it can be carried through the softened tissues with the greatest ease in almost any direction, and without causing much pain and hæmorrhage.

The discharge issuing from the sinuses differs in amount in different cases, and from time to time in the same case ; whether profuse or scanty it always exhibits the same oily, mucoid-like, slightly purulent appearance, and may stink abominably. With a very few exceptions it contains either the grey or the black grains already referred to, rarely similar bodies of a reddish or pink colour.

To the touch the swollen foot feels somewhat elastic, and does not readily pit on pressure. The sensibility of the skin is preserved. Although complained of in some instances, severe pain is rarely a prominent feature. The principal complaint is of inconvenience from the bulk and weight of the mass, and, in advanced cases, of the uselessness of the limb for locomotion. In time the foot is no longer put to the ground, different unnatural styles of progression being adopted by different patients.

As the foot enlarges the leg atrophies from disuse ; so that in the advanced disease an enormously enlarged and misshapen foot, flexed or extended, is attached to an attenuated leg consisting of little more than skin and bone. In some the tibia, or the bones of the forearm, as the case may be, become involved ; in others the disease may be at first confined to a toe, or a finger, or other limited area. In a very few instances the seat of the disease is the knee, thigh, jaw, or neck. The internal organs are never specifically implicated, either primarily or secondarily : neither are the lymphatic glands, although these may be the subject of adenitis from secondary septic infection.

After ten or twenty years the patient dies, worn out by the continued drain, or carried off more suddenly by diarrhœa or other intercurrent disease.

Classification.—Although the broad clinical features, and the progress of all cases of mycetoma are practically identical, it is customary to divide them into three varieties according to the colour of the particles suspended in the discharge; which particles, as will be presently explained, proceed from larger masses of the same coloured material lodged in the diseased tissues. Thus we have the white or ochroid, the black or melanoid, and the red forms of mycetoma. The first is the most common form, the second is somewhat less common, the third is very rare.

Morbid anatomy.—On cutting into a mycetomatous foot or hand the knife passes readily through the mass, exposing a section with an oily, greasy surface, in which the anatomical elements in many places are unrecognisable, being, as it were, fused together, forming a pale, greyish-yellow mass. The bones in parts have entirely disappeared; where their remains can still be made out the cancellated structure is very friable, thinned, opened out, and infiltrated with oleaginous material. Of all the structures the tendons and fasciæ seem to be the most resistant.

The most remarkable feature revealed by section is a network of sinuses and communicating cyst-like cavities of various dimensions, from a mere speck to a cavity an inch or more in diameter. Sinuses and cysts are occupied by a material unlike anything else in human morbid anatomy. In the black variety of mycetoma this material consists of a black or dark brown, firm, friable substance which, in many places, stuffs the sinuses and cysts; manifestly it is from this that the black particles in the discharge are derived. In the white variety the sinuses and cysts are also more or less stuffed by a white or yellowish roe-like substance, evidently an aggregation of particles identical with those escaping in the corresponding discharge. The black substance, which can be readily turned out, is moulded into truffle-like masses ranging in size from a mere grain

to a small apple, according to the capacity of the cysts or sinuses containing it. The roe-like particles in the white variety are held together by a softer cheesy-looking material. The sinuses and cysts occupy the bones, muscles, or fasciæ indiscriminately; they are found principally in the fat and connective tissue. They are lined by a smooth membrane, adherent when in the soft tissues, but capable of being enucleated when in the bones. Some of the cysts do not communicate with sinuses; most of them, however, do so and with each other, opening on the surface of the skin at the mammillated fistulæ already referred to. In the very rare red variety the colour of the accretions is red or pink. In a few instances—as in those recorded by Lewis and Cunningham—no concretions of any description can be discovered, the cysts and sinuses being occupied by an oleaginous purulent material alone.

Under the microscope mycotic elements can be discovered both in the white and black concretions. In microscopical sections of the tissues evidences of extensive degenerative changes, the result of a chronic inflammation, can be readily made out. An important feature as bearing on the pathology of the disease, and one which was long ago described by Lewis and Cunningham, has been insisted on by Cunningham (*Scient. Mem. by the Med. Off. with the Army of India*, Part 9, 1895), namely, a sort of arteritis obliterans or extensive proliferation of the endothelium of the arteries and, according to Vincent (*Ann. de l'Institut. Pasteur*, 1895), a thickening of the adventitia of the vessels as well as of the capillaries in the more affected areas.

Histology, pathology, and ætiology.—*White variety.*—The white particles suspended in the discharge from the white or ochroid variety of mycetoma are soft, easily crushed, insoluble in caustic potash, alcohol, chloroform, or acids. They are round or somewhat reniform in shape and, under the microscope, are seen to be made up of an agglomeration

of several colonies of a ray fungus—closely resembling actinomyces—in the meshes of which cellular products of inflammation are entangled.

As in actinomyces, there is a central reticulum of an exceedingly fine and interwoven mycelium surrounded by a closely set radiation of delicate, straight filaments ($1\ \mu$ to $1.5\ \mu$) many of which terminate in well-marked club-shaped ends. These clubs thus constitute the peripheral layer in a three-zoned body, the intermediate zone being the straight filaments, the central zone the interwoven mycelium. The roe-like masses in the cysts and sinuses are but aggregations of the rounded fungus group constituting the individual granule.

Sections show the same parasite in the diseased and softened tissues outside the cysts and tunnels. Thus Vincent (*Ann. de l'Inst. Pasteur*, No. 8, 1894) found it in the unbroken tuberoso swellings under the skin; Boyce and Surveyor (*Proc. Roy. Soc.*, March 9th, 1893; *Brit. Med. Journ.*, September 22nd, 1894) in the muscles and elsewhere. Kanthack (*Journ. of Path. and Bact.*, 1892-93) describes the tissue changes which it produces in this situation as follows:—"In the earliest stages we have simply a reactive inflammation, the fungus being surrounded by round cells. Gradually the latter are replaced by typical granulation tissue, epithelioid cells, and vessels which appear in large numbers. At this time the various degeneration forms of the fungus"—the clubbing of the rays, pigmentation of mycelium, vitreous changes, loss of tinctorial qualities, and so forth—"are observed, and a pigment, varying in colour from yellow to dark brown, is observed in the tissues around the fungus. Gradually the granulation cells give rise to fibrous tissue and we may then see the typical microscopical cysts or abscesses composed of the following structures passing from without inwards: (a) A fibrous ring (pigmented or not); (b) granulation cells; (c) pus cells or leucocytes, perhaps invading the fungus; (d) a finely granular detritus

immediately around the organism ; (c) the fungus itself. The latter may be at any stage of degeneration." The exact process by which the cysts and sinuses are formed has not been fully worked out ; presumably, it is an extension of the process described by Kanthack, the lining membrane of the cysts and sinuses being the outcome of an inflammation which is excited in the tissues by the fungus, and which is evidence of an effort on the part of the body to protect itself by shutting off and extruding the invading parasite.

This fungus, so closely allied to actinomyces, has been successfully cultivated by Boyce and Surveyor, and also by Vincent ; hitherto, all attempts to grow it in the lower animals have failed. Unlike actinomyces, it will not grow readily in animal substances but flourishes in vegetable infusions, or in media containing a proportion of vegetable matter. It differs also from actinomyces in certain minute morphological and tinctorial qualities ; so that though closely allied to the better-known fungus, apparently it is not specifically identical with it. Vincent has named it *Nocardia* or *Streptothrix maduræ*.

Black variety.--Although all observers are practically agreed in recognising the presence of a ray fungus in the white or ochroid form of mycetoma, there is not the same unanimity in regard to the presence and characters of fungus elements in the black or melanoid variety.

The individual black grains, floating in the discharge or lying in the tissues, in this form of mycetoma, vary in size from microscopic grains to masses of considerable bulk, as already mentioned. The surfaces of the smaller grains are distinctly mammillated, and the bodies themselves are firm and friable. Their colour is not discharged by alcohol or chloroform, and is but slightly affected by caustic potash ; but it is discharged by first boiling in caustic potash and subsequently transferring to distilled water. Certain of the grains presumably the oldest reveal no fungus

under the microscope ; but others, as Kanthack and Boyce have pointed out, show a distinct network of interlacing, broad, varicose, moniliform tubes with a manifest radiated arrangement. At the periphery these tubes may end in minute clubs. The walls of the tubes, as well as the intermediate substance, are pigmented. Such bodies do not specially suggest a ray fungus ; but Kanthack, in one specimen of melanoid Madura foot, found examples in which a central interwoven mycelium was surrounded by a zone of characteristically clubbed rays. In this specimen the filaments were broad, often interrupted, not uniform in diameter, and presented small varicosities. Transition forms, connecting the more or less perfect type with the structureless black masses, could be traced. From this circumstance Kanthack concludes that the black masses are really the product or remains of the fungus described. The black pigment is never found free in the softened tissues, but is always closely surrounded by a dense, fibrous tissue, or it lies in cysts or sinuses.

The relationship of the white to the black variety.—The question suggests itself are the two forms of Madura foot, the ochroid and the melanoid, caused by the same parasite, by varieties of the same parasite, or by a distinct species? In favour of the specific identity of the forms are the facts that black and white masses have been found together in the same case (Lewis and Cunningham and Boccaro) ; that they are co-endemic ; that, with the sole exception of the colour, the clinical features and progress of the disease are identical in both forms ; that pigment is often found in the tissues in the white variety ; that the fungus of the latter often undergoes distinct pigmentary degeneration such as is often seen in human and bovine actinomycosis. Against their identity are the rarity of their concurrence in the same individual ; the large size of the mycelial filaments in the melanoid form ; and the fact that the latter, unlike the white fungus, has hitherto resisted all attempts at cultivation.

The pathological significance of the fungus.—Most pathologists agree in regarding the ray fungus as the cause of the disease in, at least, the white variety of mycetoma. Cunningham, however, has demurred (*Sc. Mem. by the Med. Off. with the Army of India*, No. 9, 1895) to this conclusion, on the following grounds:—"1st, that a certain number of cases occur in which, whilst all the essential symptoms of the disease are present, there is an entire absence of any concretions of either the black or white variety and of the fungal elements ordinarily associated with them; 2nd, that the fungal elements which ordinarily occur in connection with the two varieties of the disease are of absolutely unlike character, those associated with the white variety resembling those of actinomyces, and those of the black variety forming sclerotinæ like those of the sclerotinæ and other allied ascomycetes; 3rd, that the very formation of sclerotoid bodies is a process which is normally indicative of a cessation of vegetative growth dependent on exhaustion of nutritive supply, and therefore one which could not occur were the normal tissues the proper site for the development of the parasitic elements." Cunningham suggests that the fungus vegetates on a soil previously prepared for it by the true, and as yet undiscovered, cause of the disease; and hints that possibly the endarteritis already referred to may be the primary effect of the true germ, and the cause of the softening of the tissues, which thus become a suitable nidus for the actinomyces like fungus that subsequently enters accidentally from without. According to this view, the ray fungus is not a necessary element in the pathological process, but merely an accidental epiphenomenon.

Until irrefutable evidence, such as can only be supplied by the successful inoculation of cultures of the ray fungus taken from both the white and the black varieties, is obtained, similar objections can always be raised to accepting these parasites as the true cause of mycetoma. Nevertheless, the intimacy

and frequency of their association with this disease are so marked that the provisional assumption of such a relationship seems to be justifiable.

Mode of entrance of the fungus.—Nothing is known on this point. It is conjectured that the fungus may be a usual parasite on some plant, and that it finds an entrance into the tissues of man through a wound in the skin. The facts of its occurring almost invariably on the feet or hands, and principally in bare-footed agriculturists, favour this view. No distinct relationship has been traced between the disease and any particular type of soil or vegetation.

Treatment.—The only effective treatment, in the case of implication of a considerable part of the foot or hand, is amputation. This must be performed well above the seat of the disease ; for it must be borne in mind that the long bones may be implicated as well as the small bones, and that unless the entire disease be removed it will recur in the stump. Complete removal is not followed by relapse. If a toe, or a small portion of the foot or hand, is alone involved, this may be excised with success. Iodide of potassium, which seems to be so potent a remedy in actinomycosis, has been tried in madura foot, but without success.

DHOBIE ITCH.

In view of the recent researches of Sabouraud and others on the ringworms of Europe, there can be little doubt that the ringworms of warm countries are attributable to a large variety of fungus forms, probably many of them derived from the lower animals. Although, in a general way, we are familiar enough with the clinical features of these ringworms, their specific germs have not as yet been very closely studied. By the lay public all epiphytic skin diseases, more especially all forms of intertrigo, are spoken of as dhobie (washer-man's) itch, in the belief, probably not very well founded, that they are contracted from clothes which

have been contaminated by the washerman. There are many sources of ringworm infection in warm climates besides the much maligned dhobie.

In the tropics, native children often exhibit dry, scurfy patches of ringworm on the scalp; and the skin of the trunk and limbs of adults is not infrequently affected with red, slightly raised, itching rings, or segments of rings, of trichophyton infection. Sometimes these rings enclose areas many inches in diameter.

Pityriasis versicolor is also very common in the tropics. It is the usual cause of the pale, fawn-coloured, scurfy patches so frequently a feature on the dark-skinned bodies of natives. On the dark-pigmented skins of negroes, Indians, and dark-complexioned Chinese, the patch of pityriasis—contrary to what obtains in Europeans and light-skinned Chinese—is paler than the healthy integument surrounding it. The pigment in the fungus (*Microsporon furfur*) and the profuse growth of the latter conceal, as a coat of yellow paint might do, the dark underlying natural pigment of the skin.

The expression *dhobie itch*, although applied to any itching, ringworm-like affection of any part of the skin, most commonly refers to some form of epiphytic disease of the crutch or axilla. There are at least three species of vegetable or bacterial parasites which in the tropics are prone to attack these situations—namely, the trichophytions or ordinary body ringworms, the *Microsporon minutissimum* of erythrasma, and the germ of the disease I have described under the name pemphigus contagiosus.

The suffering which some of the forms of dhobie itch gives rise to is often very great. In hot, damp weather especially, the various germs proliferate very actively, producing what is often a severe dermatitis. The excessive irritation leads to scratching and, very likely, from secondary bacterial invasion, to boils or small abscesses. The crutch or axillæ, or both, are sometimes rendered so raw and tender that the patient

may be unable to walk or to dress even. The irritation and itching are usually worse at night, and may keep the patient awake for hours. Even in the absence of treatment, when the cold season comes round the dermatitis and irritation subside spontaneously. The affected parts then become dry, pigmented, and scurfy, and the fungus remains quiescent until the return of the next hot weather.

Diagnosis.—The diagnosis of mycotic dermatitis is usually easily made. The festooned margin is almost conclusive. In the case of pemphigus contagiosus the characteristic blebs, the smooth, raw, or glazed surfaces, and undermined epidermic rings are usually very apparent and render diagnosis easy. When doubt exists the microscope may be necessary; but, owing to the inflamed condition of the parts, there may be much difficulty in finding fungus elements even when the case is certainly epiphytic. A negative result is, therefore, not always conclusive against ringworm.

I am convinced that many cases of dhobie itch are produced by *Microsporon minutissimum*, and that they are really inflamed erythrasma and not trichophyton ringworm. During cold weather one often sees in the site of what, during the summer, had been a troublesome dhobie itch, a brownish furfuraceous discoloration of the crutch or axilla. The same appearance I have remarked in Europe in Europeans who had suffered from dhobie itch in the East, and on examining scrapings from the parts have found *M. minutissimum* in abundance. It would seem, therefore, that during the heat and moisture of a tropical summer this generally very unirritating parasite becomes more active and excites smart dermatitis. The same may sometimes be seen in pityriasis versicolor. I believe that those cases of *microsporon-furfur* and *minutissimum*—dhobie itch are more easily cured than the trichophyton varieties.

Treatment.—After a thorough use of soap and water, the application of Vlemminck's solution of

sulphuret of calcium (1 oz. quicklime, 2 oz. precipitated sulphur, 15 oz. water, boiled together in an earthenware vessel till reduced to 10 oz.; decant the clear sherry-coloured fluid after subsidence) every night for three or four times, generally brings about a rapid cure. A tincture of the leaves of *Cassia alata* painted on, or the crushed leaves themselves well rubbed in, are equally successful. If these fail, chrysophanic acid ointment, twenty grains to the ounce of vaseline, rubbed in twice a day till a slight erythema shows at the edge of the diseased patch, is almost invariably successful. When prescribing chrysophanic acid the physician must be careful to inform the patient of its staining effect on clothes; to warn him to stop its use so soon as the erythematous ring shows; and to be careful not to apply the ointment to the face. A writer in the *Indian Med. Gaz.* (Jan., 1898) strongly recommends the application of glacial acetic acid. It cures, he affirms, with, at most, two applications. The smarting its use entails is relieved by laying a lump of ice in a handkerchief on the part. For the ringworms of the thick-skinned natives linimentum iodi freely applied, and of double strength, is the best, and a most efficient, remedy.

Prophylaxis.—The various forms of crutch dhobie itch may be avoided by wearing next the skin short cotton bathing-drawers and changing them daily, at the same time powdering, after the daily bath, the axillæ and crutch with equal parts of boric acid, oxide of zinc, and starch.

TINEA IMBRICATA* (Fig. 102).

Definition.—A form of body ringworm peculiar to certain Eastern oceanic tropical climates, produced by a *Trichophyton* (*Lepidophyton*, Tribondeau),

* *Brit. Journ. of Dermatology*. No. 39, vol. iv.: Bonnafy, *Arch. de Méd. Nav.*, Oct., 1893; Tribondeau, *Arch. de Méd. Nav.*, July, 1899.



Fig. 102. *TINIA IMBRICATA.*

[*To face p. 622.*]

and characterised by a concentric arrangement of closely set rings of scaling epidermis.

Geographical distribution.—This peculiar form of epiphytic disease is strictly confined to warm climates. It is principally met with in the Eastern Archipelago and in the islands of the South Pacific, although it has been found to extend westward as far as Burma, and northward as far as Foochow and Formosa on the coast of China. In many of the islands of the South Pacific it affects a large proportion of the inhabitants; in some islands quite one-half. There is good reason to believe that its area of distribution is gradually extending. Thus Turner and Königer tell us that it was formerly unknown in Samoa and Bowditch Islands, where it is now very prevalent. Dr. Daniels also informs me that it was introduced for the first time into Fiji by some Solomon Islanders in 1870; by 1872, he says, it had become general among the Fijians. It was recently introduced into Tahiti and rapidly spread there among the natives. We have no accounts of such a disease in Africa or America; it is probable, however, that ere long it will be introduced into the tropical parts of both of these continents. Once introduced, it spreads very rapidly in countries with a damp, equable climate and a temperature of between 80° and 90° Fahr. Very high or low temperatures and a dry atmosphere are inimical to its extension.

Symptoms.—*Tinea imbricata* is easily recognised. At first it may be confined to one or two spots on the surface of the body; usually, in a short time, it comes to occupy a very large area. It does not generally affect the soles and palms, although it may do so; nor is the scalp a favourite site. Dr. Oswald Baker, confirmed by Tribondeau, remarks that it avoids the crutch, the axillæ, and the nails. With these exceptions it may, and commonly does, sweep over and keep its hold on nearly the entire surface of the body; so that after a year or two a large part of the skin is covered with the dry, tissue-paper-like

scales, arranged in more or less confused systems of concentric parallel lines, absolutely characteristic of the disease.

An inoculation experiment readily explains the production of the scales, their concentric parallel arrangement, and the mode of extension of the patches. About ten days after the successful inoculation of a healthy skin with *tinea imbricata*, the epidermis at the seat of inoculation is seen to be very slightly raised and to have a brownish tinge. Presently the centre of this brownish patch—perhaps a quarter of an inch in diameter—gives way and a ring of scaling epidermis, attached at the periphery but free, ragged, and slightly elevated towards the centre of the spot, is formed. In a few days this ring of epidermis has extended so as to include a larger area. A second brown spot then appears at the site of the first brown spot and in the centre of the primary scaling, expanding ring. This second spot, in its turn, gives way, producing a second and similar scaling ring, which also expands, following the first ring in its extension. Later a third and fourth ring form in the same way; and so on, until a large area of skin is covered with one or more systems of concentric parallel scaling rings, which follow each other over the surface of the body like the concentric ripples produced by a stone falling into a pool of water.

The scales, if not broken by rubbing, may attain considerable length and breadth; but, of course, their dimensions are in a measure determined by the amount of friction they are subjected to. Usually they are largest between the shoulders—that is, where the patient has a difficulty in scratching himself. The lines of scales are from one-eighth of an inch to half an inch apart. The hair of the scalp is not injured.

The fungus.—On detaching a scale and placing it under the microscope, after moistening with liquor potassæ, a trichophyton-like fungus can be seen in enormous profusion. This fungus has not been cultivated, but Tribondeau, on the evidence of finding

fructification in one observation pronounces it to be a *Lepidophyton* and not a *Trichophyton*. The parasite evidently lies between epidermis and rete, and by its abundance causes the former to peel up. As the fungus does not die out in the skin travelled over, it burrows under the young epithelium almost as soon as the latter is reproduced. Hence the peculiar concentric scaling and the persistency of the disease throughout the area involved. When the scales are washed off by the vigorous use of soft soap and hot water, the surface of the skin is seen to be covered with parallel lines of a brownish colour—evidently the slightly pigmented fungus proliferating and advancing under the young epidermis.

Diagnosis.—From ordinary ringworm it is easily distinguished by the absence of marked inflammation or congestion of the rings, by the abundance of the fungus, by the large size of the scales, by the concentric arrangement of the many rings or systems of rings, by the non-implication of the hair, and, according to Dr. Oswald Baker, by the avoidance of crutch and axillæ. From ichthyosis it is distinguished by the concentric arrangement of the scaling, by the peripheral attachment of the scales, and by the presence of abundant fungus elements.

Treatment.—The best treatment for *tinea imbricata* in natives is the free application of strong linimentum iodi. Limited patches might be treated with chrysophanic acid ointment (twenty grains to one ounce) or by the inrubbing of bruised *Cassia alata* leaves. Sulphur ointment, or sulphur fumes, act very slowly and unsatisfactorily. Clothes should be boiled or burned.

Prophylaxis.—Dr. Daniels informs me that *tinea imbricata* is comparatively rare in Tonga. This circumstance the natives attribute to their custom of oiling the body. Dr. Daniels remarks that since the Fijians adopted of late years the same practice, the disease has become somewhat less prevalent among them. Cleanliness, and the immediate and active

treatment of any scaling spot, should be carefully practised in the endemic countries.

In Tahiti the use of chrysophanic acid is now general among the natives; as a consequence the disease is not so prevalent there as it was only a few years ago.

PINTA.

Definition.—An epiphytic disease characterised by peculiar pigmented patches on the skin.

Geographical distribution.—In certain districts in tropical America—especially along the river banks in Mexico, Central America, Venezuela, Colombia, Bolivia, and one or two places in Peru, Chili, and Brazil—the district between the Juciparana and the Santo Antonio rivers (Magalhães, private letter)—there occurs an epiphytic skin disease characterised by peculiar red, or blue, or black, or white piebald spotting of the skin of a part, or of the whole, of the body. The patient emits an offensive odour, sometimes compared to that of a mangy dog, or of dirty linen. Desquamation and itching of the patches are also features of the disease. It entails no constitutional disturbance and no danger to life. Like other epiphytic diseases, want of personal cleanliness has a great deal to do with the prevalence of pinta in the districts mentioned, for it is rare in cleanly whites or well-to-do negroes; the dirty Indians and the poor half-castes are those most frequently affected. In some districts it occurs in nearly a tenth part of the inhabitants, in others nearly the entire population is affected. Lately a somewhat similar disease has been seen in North Africa (Legrain, *Arch. de Parasit.*, Jan., 1898.)*

* It is difficult from his description to determine the exact nature of the disease alluded to by Legrain. It commences with pronounced fever lasting for one week, and is followed by malaise persisting for several weeks. This is followed by itching and, by-and-by, by furfureous desquamation of the itching parts and gradually developed achromia. He positively affirms the disease is not ordinary vitiligo. He also says that he has seen in Tripoli a coloured skin affection with the clinical features of true

Pinta commences at one or two points, the rest of the surface becoming infected in turn by extension or by auto-contagion. In the first instance the hands, or face, or some other exposed parts, are attacked. The original patch may be white, red, blue, or black. It gradually increases in size, becoming scurfy and itchy, particularly when the surface is warm. As the patches spread they assume a variety of shapes. Fresh spots appear in the neighbourhood of the parent spots, into which, in course of time, they tend to merge; so that ultimately large patches of discoloured skin are formed. The palms of the hands and the soles of the feet are not attacked. On the scalp becoming affected the hair turns white and thin, and ultimately falls out. When fully developed, the disease produces a very grotesque appearance. It is probable that the white patches in a proportion of cases are not epiphytic, as they neither itch nor desquamate; very likely they are ordinary leucoderma, brought about by disturbance of the natural pigmentation of the skin by a parasite which had subsequently died out. Sensation and the glandular functions of the skin are not affected. In consequence of the scratching, the implicated parts may become cracked or ulcerated.

Two types of the disease have been named—the superficial epidermic and the deep epidermic; the former being represented by black and blue patches which spread rapidly; the latter including the red and white patches, apparently involving the rete and deeper layers of the epidermis, spreading more slowly, and, at the same time, being more difficult to cure. The various forms and colours may concur in the same individual; but a given patch, once established, does not change colour.

pinta, occurring in little epidemics in particular houses. The results of microscopical examinations of scrapings of the affected skin were negative. Possibly this is the disease referred to in the *Journ. of Trop. Med.*, Nov., 1899, by Sandwith, as having been seen by him in Egypt.

Pinta is contagious, and attacks both sexes and any age. Unless properly treated it may last for years.

Pathology.—If one of the scales is moistened with liquor potassæ and placed under the microscope, black spores and a white, highly refracting mycelium are found. The spores are round or oval, measuring 8μ to 12μ in diameter. Abundant pigment is seen floating in a yellowish fluid in the interior of the spore. The mycelial filaments are short, non-branching, tapering from a broad base to a blunt point by which each filament is attached to a single spore, like the stalk to a cherry. The mycelium measures from 18μ to 20μ in length by 2μ in breadth. The differences in the colour of the patches probably depend on differences in the pigmentation, or kind, of the fungus. Such is Gastambide's description of the parasite, a description which, to some extent, is borne out by Dr. Osborne Browne in the *Journ. of Trop. Med.*, Jan., 1900.

Montoya y Florez (*Recherches sur les Caratés de Colombie*, 1898) has published an elaborate and careful description of the disease and of the various mycotic growths he found in the several varieties of pinta which he has studied. He says that he has never seen a fungus answering to Gastambide's description. On placing the scales moistened with liquor potassæ under the microscope long, dichotomous filaments, generally very fine and cylindrical, in certain conditions granular and beaded, are seen. In places this mycelium forms a dense network. Here and there veritable ropes of mycelium are visible, or broad, short filaments with fructification characteristic of the particular variety of the disease may be detected (Fig. 103). He has succeeded in cultivating the various fungi which apparently belong to a plurality of genera—*Penicillium*, *Aspergillus*, *Monilia*.

Diagnosis.—This disease is readily diagnosed from leprosy by the absence of anæsthesia in the patches, and by the colour of the spots; from

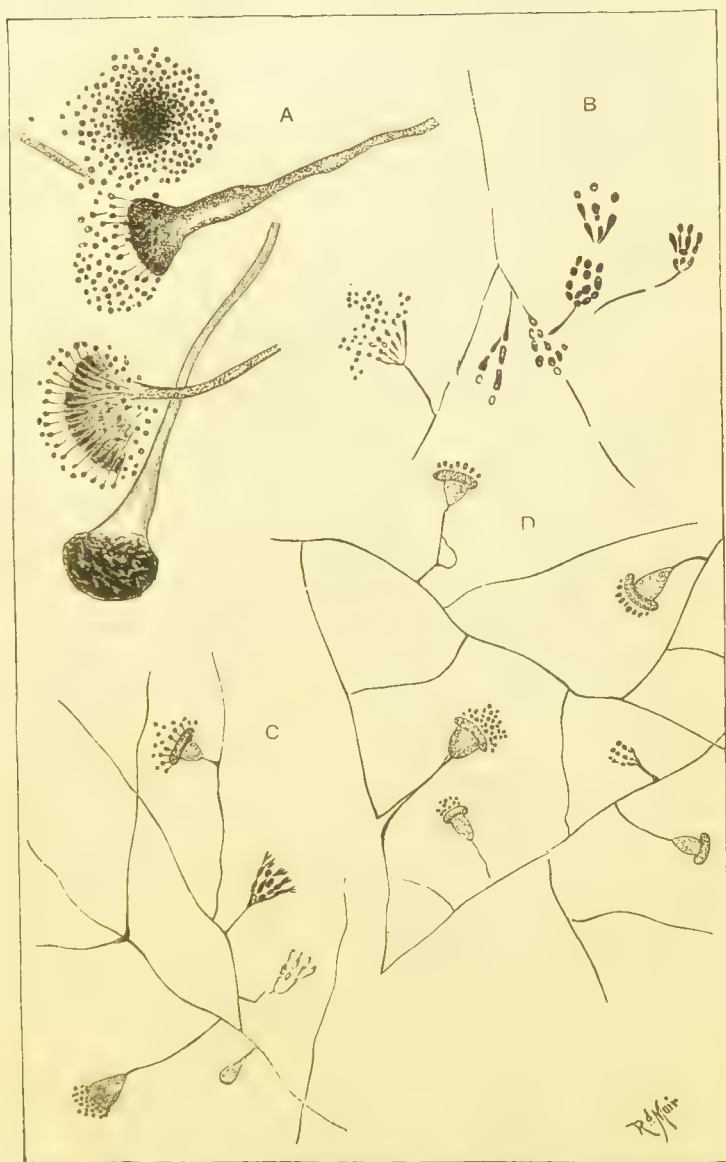


Fig. 103.—FRUCTIFICATION OF CRYPTOGRAMIC EPIPHYTES IN PINTA.
(After Montoya y Flores.)

A, Red pinta ; B, dark violet pinta ; C, grey violet pinta ; D, blue pinta.

erythrasma, from ringworm, and from pityriasis versicolor by the colour, and by the microscopical characters of the fungus.

Treatment.—Chrysophanic acid, preparations of sulphur, strong liniment of iodine, and other epiphytides are indicated. Cleanliness and the destruction of old clothes are indispensable.

PIEDRA.

This peculiar disease of the hair is very common in certain districts of Colombia, South America. So far as is known, it is confined to the inhabitants of that country, of whom a considerable proportion, both male and female, and apparently belonging to all the races represented there, are affected.

According to Juhel-Rénoy (*Ann. de Derm. et Syph.*, Dec. 25th, 1888), whose observations practically coincide with the earlier accounts by Desenne, Cheadle, Morris and others, the hairs of the affected scalp are dotted over at irregular intervals with numerous—twenty-three in a hair sixty centimetres in length—minute, gritty nodosities. These are barely visible to the naked eye, but distinctly perceptible to the touch when the hair is drawn between finger and thumb. The affected hairs are bent and twisted, and tend to produce matting and knotting. The little nodosities, which, though very firm, are not so hard as the name piedra (a stone) would indicate, being easily cut through with a sharp knife or scissors, are paler than the hair which they surround, or partly surround, like a sheath. When a comb is drawn through the hair a sort of crepitation is produced, doubtless by the friction against the hard particles.

Under the microscope these excrescences are found to consist of a number of spore-like bodies, easily made apparent by soaking the hair in liquor potassæ after washing in ether. The spores (which are twice the size of trichophyton spores and remarkably refringent) from mutual pressure are polyhedral, and

together form a sort of tessellated mosaic, the elements of which seem to be held together by a greenish soluble cement in which a number of minute bacteria-like rods are incorporated. The shaft of the hair is not eroded or affected in any way; it can be seen intact through the encrusting fungus.

Piedra is supposed by some to be induced by the mucilaginous hair applications in vogue among the Colombians. Although Juhel-Rénoy has given to it the name "trichomycose nodulaire," it must not be confounded with the trichomycosis nodosa of Paterson (the leptothrix of Wilson), which is quite a different affection and common enough on the axillary, scrotal, and face hair in Europe and elsewhere. Neither must it be confounded with trichorexis nodosa, a non-parasitic disease of the hair-shaft, which is split up at different points into brush-like bundles of fibres and is thus easily fractured; nor with moniliform hair (monilethrix, Crocker), a congenital, hereditary, and also non-parasitic disease.

Treatment.—Cleanliness, the free use of soap, and the application of some epiphyticide should suffice for cure. Should such means fail, doubtless shaving the scalp would be effectual.

IV.—*Caused by animals.*

THE CHIGGER OR SAND FLEA (*Pulex penetrans*).

This insect, formerly confined to the tropical parts of America (30° N. to 30° S.) and to the West Indies, appeared on the West Coast of Africa for the first time about the year 1872. Since that date not only has it spread all over the tropical parts of that continent but, according to



Fig. 104. — Chigger (*Pulex penetrans*).
(Blanchard.)

Blandford (*Entomologists' Monthly Mag.*, May, 1894), has made its appearance in China. As a cause of suffering, invaliding, and indirectly of death, it is an insect of some importance. It is now extremely prevalent on the East Coast of Africa, and is causing a large amount of invaliding amongst the Indian coolies there, by whom, it is to be feared, it may be introduced into Bombay.

The chigger (Fig. 104) is not unlike the common flea (*Pulex irritans*) both in appearance and, with

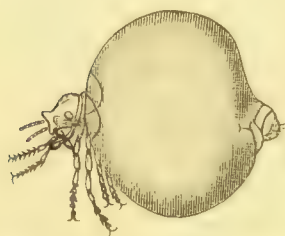


Fig. 105.—Chigger; impregnated female. (Blanchard.)

one exception, in habit. It is somewhat smaller in size, the head being proportionately larger and the abdomen deeper than in the latter insect. In colour it is red or reddish brown. Like the flea, its favourite haunt is in dry, sandy soil, the dust and ashes in badly kept native huts, the stables of cattle, poultry pens, and the like. It greedily attacks all warm-blooded animals, including birds and man. Until impregnated, the female, as well as the male, is free, feeding intermittently as opportunity offers. So soon as she becomes impregnated she avails herself of the first animal she encounters to burrow diagonally into its skin, where, being well nourished by the blood of the host, she proceeds to ovulation. By the end of this process her abdomen, in consequence of the growth of the eggs it contains, has attained the size of a small pea. (Fig. 105.) The first anterior and the two posterior segments do not participate in the enlargement, the latter acting as a plug to the little hole made by the animal on entering the skin. When the ova are mature they are expelled and fall on the ground. In a short time a thirteen-ringed larva is hatched out from each egg. This larva presently encloses itself in a cocoon, from which, in eight to ten days, the perfect insect emerges.

During her gestation the chigger causes a considerable amount of irritation. In consequence of this pus forms around the distended abdomen, which now raises the inflamed integument into a pea-like elevation. After the eggs are laid (according to some,



Fig. 106.—CHIGGERS IN SOLE OF FOOT.

(From a photograph by Dr. Daniels.)

before this process) the superjacent skin ulcerates and the chigger is expelled, leaving a small sore which, if infected by any pathogenic micro-organism, as the bacterium of phagedæna or of tetanus, may lead to grave consequences.

Naturally, being nearest the ground, the feet are the parts most commonly infested by chiggers. The soles of the feet (Fig. 106), the skin between the toes, and that at the roots of the nails are favourite situations.



Fig. 107.—Chigger lesions of hands and feet.
(From a photograph by Dr. Daniels.)

Other parts of the body are by no means exempt ; the scrotum, penis, the skin around the anus, the thighs, and even the hands (Fig. 107) and face are often attacked. Usually only one or two chiggers are found at a time ; occasionally they are present in hundreds, the little pits left after their extraction being sometimes so closely set that parts of the surface may look like a honeycomb.

Treatment.—In chigger countries, houses, par-

ticularly the ground floors, must be frequently swept and accumulation of dust and *débris* be prevented. The housing of cattle and poultry must be similarly attended to. The floors should be sprinkled often with carbolic water, insect powder, or similar insecticide. Walking barefooted must be avoided. Bathing must be practised daily, and any chiggers that may have fastened themselves on the skin at once removed. They may be killed by pricking them with a needle, or by the application of chloroform, turpentine, mercurial ointment, or similar means, after which they are expelled by ulceration. The best treatment, however, is not to wait for ulceration, but to enlarge the orifice of entrance with a sharp, clean needle and neatly to enucleate the insect entire. Some native women, from long practice, are experts at this little operation. The part must be carefully dressed and protected until whole.

MYIASIS.

THE SCREWORM (*Lucilia macellaria*).

This is the larva of a dipterous insect (Fig. 108) common in certain parts of America, from the United States to the Argentine. The insect lays her eggs on

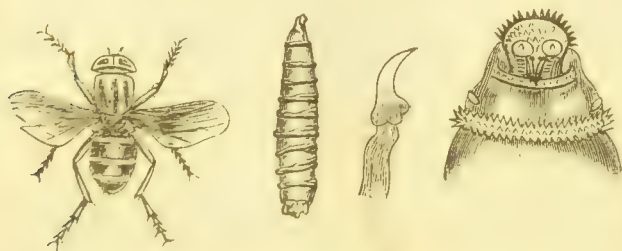


Fig. 108.—Screwworm. (Blanchard.)

the surface of wounds, and in the ears and nasal fossæ of persons sleeping in the open air. From these eggs the larvae are hatched. The latter are white, about three quarters of an inch in length, and formed of twelve segments carrying circles of minute spines so

arranged as to give the creature a screw-like appearance; hence the vulgar name. They burrow in the tissues, devouring the mucous membrane, muscles,



Fig. 109.—Native with *Lucilia macellaria* in nostrils and frontal sinuses: early stage. (From a photograph by Dr. Daniels.)

cartilages, periosteum, and even the bones, thereby causing terrible sores, and not infrequently, particularly when they attack the ear or nasal fossæ, by penetrating to the brain, death. (Fig. 109.) In thirteen cases collected by Laboulbène nine proved fatal: in thirty-one collected by Maillard twenty-one died.



If treated properly and in time by injections of chloroform, carbolic acid, turpentine, and similar substances, the patient may be saved; neglected, he will most probably die. If necessary the frontal sinuses, the antrum, and other bony cavities must be opened to secure the expulsion of the larvæ. In countries where this pest occurs bloody and offensive discharges from the nostrils should be carefully investigated, and if found to be caused by the screwworm vigorously treated.

VER MACAQUE (*Dermatobia noxialis*) (Fig. 110).

This is the larva of an American fly the identity of which, until it was studied by Blanchard (*Bul.*

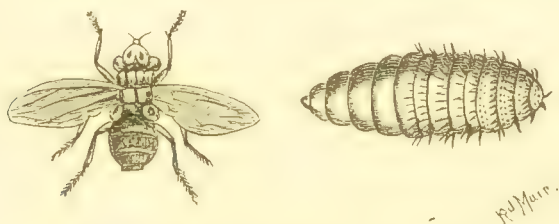


Fig. 110. — *Dermatobia noxialis*. (Blanchard.)

de la Soc. Cent. de Méd. Vétérinaire, July 9th, 1896), was rather doubtful, some naturalists considering that it might be the larva of *D. cyaniventris*. Blanchard has now shown that the latter never attacks man or domestic animals. Though *D. cyaniventris* is somewhat larger than *D. noxialis*, it is much less formidable. *D. noxialis* occurs about wooded lands, and deposits her eggs on cattle and dogs and occasionally on man. When hatched out the larvæ penetrate the skin and produce an inflamed swelling about the aperture of entrance, from which a sero-purulent fluid, containing the black fæces of the larva, exudes. Depied (*Arch. de Méd. Nav.*, February, 1897) says that he has twice encountered this larva (the identity of which he ascertained by developing the insect) in the scalp of Tonquinese.

VER DU CAYOR (*Ochromyia anthropophaga*).

This insect and larva occur in the district of Cayor, Senegambia. The larva burrows into the skin and produces a small inflamed swelling, from which it emerges in from six to seven days.

In Africa, and in many other parts of the tropical world, similar anthropophagous larvæ, which, however, have not yet been satisfactorily identified, are frequently encountered.

IXODIASIS.

The numerous observations which in recent years have established the fact that many of the blood parasites of vertebrates habitually employ blood-eating arthropodes as intermediary hosts, either for the completion of their development or for passage from host to host, countenance the notion that those essentially blood-eating animals, the ticks, may fill a similar rôle. Indeed, this has already been proved as regards the parasite (*Pirosoma bigeminum*) of Texas cattle fever (p. 4), which, as originally shown by Smith and Kilborne and confirmed by Koch, after being abstracted by the tick (*Rhipicephalus sanguineus* v. *Boophilus boris*) passes into the egg, and so into the young of this acarus by which, when the acarus comes to feed, it is in turn introduced into the body of another bovine. Certain imperfect observations in respect of two species of tick, *Acarus persicus* and *Acarus moubata*, point to a similar transmission of pathogenic organisms by them to man. In the hope that by calling attention to the subject something more definite may be elicited, I have condensed the little that is known about the pathological effects sometimes, though not invariably, produced by the bites of these animals. The facts that these bites are not invariably followed by serious consequences, that when serious consequences do ensue these are not evanescent, and that they confer immunity, suggest that something more than simple

poisoning by an animal secretion occurs, most likely something of the nature of the introduction of a living germ.

The ticks, or wood-lice, are allied to the spiders, and belong to the order *Acarus*, of which they are the largest specimens. They are all of them parasitic on land vertebrates, most of them having special zoological distributions, although each may have a limited variety of hosts. Consequently most species have a restricted geographical range; a few, being parasitic on migratory birds and on domestic animals, are more cosmopolitan.

Briefly, their life-history is as follows:—The newly-hatched *larva* possesses only six legs, and breathes through stigmata placed behind each pair of limbs. The *larve* ascend plants, from which they transfer themselves to any passing animal. If this prove a suitable host the ticks attach themselves to its skin by means of their beaks for two or three days. After thus feeding they fall off and, lying concealed in the ground, undergo a first metamorphosis, during which they acquire a fourth pair of legs and additional stigmata. Thus furnished, the *pupa* ascends some plant, again feeds on a vertebrate host, and once more drops on the ground to undergo a final metamorphosis and acquire sexual organs. After impregnation, the male dying, the female finds her host, fills herself with blood, and, dropping off, lays many thousands of eggs. The life-span of the individual tick depends on circumstances. If unfed it may remain alive, though inactive, for several years.

ARGAS PERSICUS.

Several travellers have reported on a peculiar disease, especially common in Miana in the north of Persia, which the natives confidently attribute to the bite of *A. persicus*. According to Kotzebue and Waldheim, the bite of this tick is followed by severe pain, delirium, convulsions, and sometimes even by death. Schlimmer says the symptoms resemble those “of remittent fever—extreme lassitude, disinclination to work, yawning, fever, perspiration not accompanied by much thirst, increasing and decreasing at stated hours in the day.” Fatigued travellers and those who have undergone privations are specially liable. He relates that on one occasion (1858) he treated 400 soldiers who declared they had

been bitten by these ticks at Miana. Natives, he says, are immune, a circumstance he explains by previous attacks. According to Dupré, the Persians treat the disease by interdicting meat, acid or fermented drinks, and by the administration of sugar, which they regard as a specific.

Argas persicus is found in the north of Persia, where it is called Guérib-guez, Malleh, and Keuch bheb-guez. Its habits are similar to those of the common bed bug, infesting old houses, living in the cracks of walls, floors, or furniture, and coming out at night to feed on men or beasts. Kotzebue says that it may so infest a village as to drive away the inhabitants.

Megnin, because he experienced no ill effects from the bite of a four-year-old *A. persicus*, denied the pathogenic properties of this tick. Tholazan, however, who while in Persia paid special attention to the point, believes that its evil reputation is well deserved.

ORNITHODORUS MOUBATA (*Argas moubata*, Murray).

Dr. Livingstone was the first to describe the tick disease of Portuguese South Africa. He met with it at Ambaca and Tete on the Zambesi. He says, "the effects of the bite are a tingling sensation of mingled pain and itching, which gradually ascends the limb until it reaches the abdomen, where it soon causes violent vomiting and purging. When these effects do not follow, as we found afterwards at Tete, fever sets in; and I was assured by intelligent Portuguese there that death has sometimes resulted from this fever."

Later Sir John Kirk referred to the same disease, which he found in the Zambesi valley as high up as Sescheke, above Victoria Falls. "The symptoms," he says, "appear soon after the bite and are sharp fever, vomiting and often delirium; in about two days these pass off, but there is no marked profuse perspiration as in malarial fever. After recovery the

patient has complete immunity from further attacks however he may be bitten, but it is doubtful whether this immunity lasts for any length of time in case of removal."

Dr. Dowson, R.N., in a letter accompanying specimens of the tick sent to the British Museum, says, "the Portuguese believe that the severity of the subsequent symptoms depends on the number of bites received. Severe fever and dysentery, with great swelling around the anus, are stated to have occurred. . . . A Dutch trader assured me that he was bitten by one of these *acari* on the toe and that the bite was very painful. A few hours later he was seized with most violent abdominal pain, vomiting and purging. The stools contained blood. He was ill for three days afterwards, but said there was no fever at all."

Dr. Daniels, writing to me from British Central Africa, says, "I have come across tick fever. Usually several ticks must bite; they produce a small lump, which remains for two or three days. In from five to ten days the subject of the bites gets an attack of abdominal pain with vomiting and purging. The stools are somewhat dysenteric. This lasts for nearly a week and is accompanied by fever and rigors. After that fever continues often for three or four weeks. Some people are not susceptible; others get only one attack; a few may have several, but in time they also become immune. The case I saw, an adult, was in the second week of the illness and the purging had stopped. The temperature ranged from 100° F. to 102.3° F. Three blood examinations were negative. There were no physical signs."

In Tete the tick is called *Kufu* or *Bu* by the natives, *Tampan* or *Garrapato* by the Portuguese.

Mr. Pocock, the distinguished arachnologist of the British Museum, considers the Tete tick to be identical with *Ornithodoros moubata* found by Welwitsch in Angola. It has been confounded with *O. savignyi* of Egypt, Somaliland and British East Africa, but it

differs from this allied but non-pathogenic species in several minute anatomical details. *O. moubata* extends across Africa along the Zambesi valley and its tributaries. In habit it resembles the common bed bug, it inhabits old houses, hiding during the day in cracks and moving about actively during the night in search of the blood of man or beast. In some districts the natives protect themselves from the pest by plastering the walls and floors of their huts with mud and cow-dung, a practice adopted by the Boers, the Bechuanas and nearly all the cow-keeping native tribes. They frequently smoke their huts to drive the ticks from their lodgment in the thatch. The Portuguese at Tete have a great dread of the Garrapato, and always warn new arrivals not to place their beds on the ground, and to be careful to have the inside of their mosquito nets carefully searched for the tick before retiring for the night.

LEECHES.

In the grass and jungle lands of many tropical and subtropical countries land leeches, probably of several species, often occur in great abundance ; so much so that in some circumstances they may prove to be something more than a nuisance. The *Hemadipsa ceylonica* is one of the most active, as well as best known, of these. Before feeding it is about an inch in length and about the thickness of a knitting-needle. It clings to a leaf or twig awaiting the passing of some animal, on which it springs with remarkable activity ; it at once attaches itself to the skin, and proceeds to make a meal on the blood. Animals are sometimes killed in this way ; men even have been known to succumb to the repeated small bleedings by these pests. It is necessary, therefore, when passing through jungle lands in which leeches abound to have the feet and legs carefully protected. The bite is not infrequently the starting-point for a troublesome sore.

In the south of Europe and in the north of Africa

the horse-leech, *Hæmopsis sanguisuga*, sometimes gets into the gullet and nostrils of men as well as of animals. It has occasionally caused death by entering and occluding the air-passages. In Formosa I heard of and saw several instances of a similar form of parasitism, both in men and monkeys. To what particular species the leech in these cases belonged I do not know. Doubtless when very young they were taken in unperceived with foul drinking water, and, wandering around the soft palate, found their way into the nose. Occasionally, in the cases I refer to, the animal would protrude from the nares and wander over the upper lip. For a long time they contrived to elude all attempts at capture. By dipping the face in cold water they could generally be persuaded to show themselves. In one instance the leech dropped out spontaneously. In another—an American naturalist who had been travelling much in the interior of Formosa, and who had suffered from severe headache and profound anæmia, the result of repeated epistaxis—I succeeded in removing the leech by attaching through a speculum a spring forceps to its hinder end, and afterwards injecting salt and water. It would be well, therefore, to bear in mind that in tropical countries persistent headache, associated with recurring epistaxis, may be caused by a leech in the nostril.

SECTION VII.--LOCAL DISEASES OF UNCERTAIN NATURE.

CHAPTER XL.

CLIMATIC BUBO—GOUNDOU—AINHUM.

CLIMATIC BUBO.

SCHEUBE (*Arch. für klinische Med.*, vol. 64, translated in *Journ. of Tropical Medicine*, June-July, 1899) has applied the term climatic bubo to a type of non-venereal adenitis not uncommon in tropical climates. So far as available statistics show, the disease is specially prevalent among the crews of war-ships on the eastern coast of Africa. It occurs also in the Straits of Malacca, in China, where I have seen a fair number of cases both in landsmen and in sailors, in the West Indies, Japan, the Mediterranean and, probably, in many other places, including, perhaps, in a minor degree, Europe. It appears to be epidemic at times in certain places, and to prevail in groups of individuals living under similar hygienic conditions. Thus, Ruge reports thirty-eight cases in the German squadron blockading the Zanzibar coast in 1888-89; Godding (*Brit. Med. Journ.*, September 26th, 1896) notes its frequency in the British fleet, also on the East African coast; and Skinner (*Brit. Med. Journ.*, January 9th, 1897) mentions forty-nine cases which occurred in a regiment and battery of artillery, twenty-eight in Calcutta, thirteen in Hong-kong, four in England, two in Allahabad, and two in Malta.

The disease generally commences with fever of a remittent type in association with inflammatory

swelling, usually of a sub-acute character, of the groin glands. The oblique inguinal glands are those most frequently affected, but at times it is the crural glands that are attacked. Sometimes both groins are affected, sometimes only one, sometimes one groin is attacked after the other. The affected glands slowly or more rapidly enlarge to the size of a hen's egg, or even larger. After several weeks, it may be months, the swelling slowly subsides. Occasionally the periglandular connective tissues inflame, the integuments become adherent, and suppuration ensues. If the suppurating glands be freely incised, or excised, the parts readily heal; but if they are left alone, or inefficiently treated, fistulous tracks form and may take a very long time to heal.

Hitherto no satisfactory explanation of this type of adenitis has been forthcoming. No special bacterium has been demonstrated in the tissues. There are no adequate reasons for supposing, as has been conjectured, that the disease has any connection with plague, or that it is a form of pestis minor. Most probably the adenitis depends on some virus which has been introduced through an overlooked wound or insect bite on the legs or genitals.

Treatment should consist in rest and soothing applications during the more acute stage. After pain and tenderness have subsided graduated elastic pressure should be applied. Concurrent malaria would call for quinine; syphilis—for mercury or the iodides.

GOUNDOU OR ANAKHRE (*Gros Nez*) (Fig. 111).

On December 10th, 1882, Professor A. MacAlister read a paper before the Royal Irish Academy on what were termed the horned men of Africa. In the *British Medical Journal* of December 10th, 1887, Surgeon-Major J. J. Lamprey gave further details, illustrated with drawings, on the same subject. He had seen three such cases on the West Coast of

Africa, all of them Fantees; one came from the Wassan territory, one from the Gamin territory, the third was a visitor to Cape Coast Castle. Dr. W. Renner (*Journ. Trop. Med.*, Jan., 1900) also reports and illustrates a case from the Sierra Leone River.

In the *Archives de Médecine Navale*, January, 1895, Maclaud calls attention to what is manifestly the same affection, which, according to him, occurs



Fig. 111.—Goundou or Anakhre. (Maclaud.)

in a considerable proportion—one or two per hundred—of the inhabitants of certain villages on the Ivory Coast. The natives call it *goundou* and also *anakhre*. Maclaud says it is confined to the riverain districts of the lower Camoe; according to the information he received, if found elsewhere it is only in individuals who had previously resided in this district. Lamprey's and Renner's observations prove that *goundou* has a considerably wider distribution.

According to Maclaud, the disease usually commences soon after childhood, although adults may also be attacked. The earliest symptoms are severe and more or less persistent headache which, after a time, is associated with a sanguino-purulent discharge from the nostrils, and the formation of symmetrical

swellings the size of a small bean at the side of the nose. Apparently the swelling affects the nasal



Fig. 112.—Goundon in a West Indian Child. (*Dr. Henry Sturges.*)

process of the superior maxilla. The cartilages are not involved. Although Maclaud does not refer to

this point, it may be assumed that the nasal ducts remain patent. After continuing for six or eight months, the headache and discharge subside. Not so the swellings; these persist and continue slowly and steadily to increase until in time they may attain the size of an orange, or even of an ostrich's egg. As they grow, the tumours, encroaching on the eyes, may interfere with the line of vision and finally destroy these organs. There is no pain in the tumours themselves. The superjacent skin is not involved, being healthy-looking and freely movable. The tumours are oval, with the long axes directed downwards and slightly from within outwards. Lamprey's drawings give a more elongated form and horizontal direction. They look, according to Maclaud, when of moderate dimensions, something like two half-eggs laid alongside the nose, one on each side. The nostrils are bulged inwards, and more or less obstructed; but, in the later stages at all events, there is no discharge, neither can any breach of the mucous surface be detected. The hard palate is not affected in any way.

Maclaud had no opportunity of ascertaining by *post-mortem* examination or by surgical operation the nature of this singular disease. He inclines to the opinion that, in the first instance, the process is started by the larvæ of some insect which find their way into the nostrils. I would point out, however, that the symmetry of the growths is difficult to account for on this hypothesis. Maclaud observed a similar affection in a chimpanzee.

Dr. Henry Strachan (*Brit. Med. Journ.*, Jan. 27th, 1894) records and illustrates (Fig. 112) an instance of what may be the same affection in a West Indian negro child. In this case the swellings were congenital, and had only increased in proportion to the child's growth. They were hard, smooth, bony masses, somewhat of the shape and size of an elongated pigeon's egg, and sprang from the nasal process of the superior maxillary and nasal bones. For



In the Ann. of Trop Med. 15th Dec. 1900, Dr
Lester ^{and separately} ~~marked~~ ^{describes} a well marked
example of gonorrhea in a Chinese woman, etc
in whom the disease began when she was a
girl and steadily progressed for some years
and then became stationary

æsthetic reasons they were removed by the chisel, and were found to consist of compact bone with a cancellous core. Dr. Strachan states that he had seen two similar cases, and had often noted a "ridge" in this part of the face of West Indian negroes. He suggests that the condition may be an example of atavism, referable to some tribal peculiarity of the original West African stock.

Dr. Chalmers, Assistant Colonial Surgeon, Accra, has given (*Lancet*, January 6th, 1900) an admirable and well-illustrated account of this affection as seen on the Gold Coast, where it is fairly common and is known as "henpurge." He confirms Strachan as to the anatomical characters of the swellings, which he regards as the result of an osteoplastic periostitis due to yaws. He affirms that the morbid process begins during, or soon after, an attack of yaws, and is correlated in some way to an anatomical arrangement of the blood-vessels of the parts, an arrangement which, he gives the reader to infer, is peculiar to the negro of this part of Africa.

AINHUM (Fig. 113).

This is a disease of a very peculiar character, affecting the toes, particularly the little toes, of negroes, East Indians, and other dark-skinned races.

It commences as a narrow groove in the skin almost invariably on the inner and plantar side of the root of the little toe. It may occur on one foot only, or in both feet simultaneously, or it may affect one foot after the other. The groove, once started, deepens and extends gradually round the whole circumference of the toe. As the groove deepens—it may be, though not necessarily, with some amount of ulceration—the distal portion of the member is apt to swell to a considerable size, as if constricted by a ligature. There is little or no pain, although there may be inconvenience from the liability to injury to which the dangling and now everted toe is exposed. In the course of years the groove slowly deepens and finally

the toe drops off or is amputated. The groove may either correspond with a joint or it may be formed over the continuity of a phalanx. In rare instances, after the two distal phalanges have dropped off or been amputated, the disease recurs in the stump and the proximal phalanx in its turn is thrown off. Of



Fig. 113.—Ainhum.

the other toes the fourth is the one which is most frequently affected; very rarely are the third, or second, or great toes attacked. In the Army Medical Museum at Washington, U.S.A., there is a wax model representing a case of this, or a similar affection, in which all the toes had been thrown off and the disease was making progress in the leg. Ainhum is very rare in women or children, being most common in adult males. It runs its course in from one to ten or even more years.

On section it is found, as a rule, though not invariably, that the panniculus adiposus of the affected toe is much hypertrophied, that the bone is infiltrated with fatty matter, and that the other tissues are correspondingly degenerated. Sometimes the bone is thinned, or even altogether absorbed. At the seat of constriction a line of hypertrophy of the epithelial layers, and of atrophy of the papillary layer of the skin, together with a band of fibrous tissue more or less intimately connected with the derma, surround, in whole or in part, the narrow pedicle.

Nothing is known as to the true nature and cause of this disease, to which the European and white-skinned races are not, but to which the African races, particularly the negroes of the West Coast, are specially liable. Some have suggested that it is a trophic lesion depending upon some nervous affection. The occurrence of severe loin pains, which Dupouy says he remarked at the commencement in some of his cases, as well as the tendency of the affection to run in families, as noted by Da Silva Lima, affords a certain amount of support to this view. Others suggest that it is a manifestation of leprosy; others that it is a form of sclerodermia; others, again, and on equally inadequate grounds, that it is produced artificially by intentional ligation or by the wearing of toe rings. My own impression is that it is provoked, at all events in the first instance, by wounds—so easily inflicted on bare feet in walking through grass or jungle. The fold of skin in which the lesion of ainhum commences is very liable, especially in the splayed-out toes of the negro, to be wounded in this way. If we examine the under surface of the joint flexures of the toes in many individuals of this race, even of those not affected with ainhum, we often find the skin, particularly at the proximal joint of the little toe, thick, rough, scaling, and sometimes even ulcerated. One can understand that continual irritation of this sort, produced and kept up by wounds from sharp grasses and so forth, would in time give

rise, especially in the dark-skinned races so prone to keloid, to fibrotic changes in the derma, which might very well end in a sort of linear cicatricial contraction and ultimately in slow atrophying strangulation of the affected member. The disease is said, however, to have been seen in those who wear shoes; but, unless it could be shown that such individuals had always worn shoes, this objection to the explanation offered would not apply. I have seen a negro in whom the entire integument of the little toe was involved in a sclerodermia, and the part in consequence was shrunk and hide-bound, whilst the little toe of the other foot was affected with well-marked ordinary ainhum; the process was diffuse, as it were, on the one side, localised on the other.

The tail in certain species of monkey is liable to a similar disease. I have had under observation for some time a pet monkey in which the part corresponding to the distal vertebra dropped off in consequence of an ainhum-like linear constriction. Two months later the next vertebra was similarly amputated, and now a third groove is forming a little higher up the tail.

Treatment.—It has been suggested that division of the constricting fibrous band would delay the evolution of the disease. In the early stage this might be tried. When troublesome the affected toe should be amputated.

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